ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 55 JUNE, 1946 No. 2

XXI

A BRIEF REVIEW OF RHEUMATIC FEVER. THE ROLE OF TONSILLITIS IN ITS ETIOLOGY.

DAVID HIGBEE, M.D.

SAN DIEGO, CALIF.

Although rheumatic fever is of first importance in the practice of internal medicine and pediatrics, it concerns otolaryngologists because acute upper respiratory infections so commonly precede the onset of the disease.

During the past few years military life has offered exceptional opportunities for the study of rheumatic fever in the adult. Much of its life cycle, remote effects, epidemiology and distribution are now known for the first time. Previously this disease has been considered one of childhood, but it is now known that passing the second decade is no assurance of immunity. In some branches of the services there was an incidence of one per thousand new cases.

It is, nevertheless, in children that we will most frequently encounter it. Ninety per cent of cases occur under the age of 15. An otolaryngologist may be the first to see rheumatic fever in its early stages, because a bewildered parent may only remember that the lingering illness of his child began some weeks before with a fever and sore throat. We shall also see this disease in consultation

Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, Calif., Jan. 27, 1946.

with the pediatrician who will want advice about the presence of chronic infection, and desire us to share with him the responsibility for dealing with it.

Of all the conditions which develop in childhood and tend to become chronic, rheumatic fever is by far the most serious problem. In the five to ten year age group this disease is second only to the acute contagious diseases as a cause of death. Between the ages of ten and fifteen its mortality is higher than for any other disease. Each year in the United States approximately 170,000 new cases develop. Because of its tendency to become chronic and produce cardiac damage, 840 of each 100,000 population are afflicted with rheumatic heart disease.[2]

A statistical study of the life cycle of rheumatic disease was published by Cohn and Lingg[1] in 1943. The report covered 3129 patients with rheumatic carditis which were followed over a period of 15 years. Fifty per cent of these patients had died within 9 years; 25 per cent lived more than 17 years; 10 per cent lived for 30 years. The average age at the time of the initial attack was 15 years.

Rheumatic fever develops only in the presence of a number of factors which seem equally important. The hemolytic streptococcus and particularly group A is almost universally present either in the nose, nasopharynx, or tonsils.[5] This organism has been cultured frequently during the month preceding the first symptoms of the disease and is generally present during the initial attack. The fact that only a small percentage of individuals who show positive cultures of this organism develop rheumatic fever gives added emphasis to other factors.

In the past climate has been considered a major factor. The great majority of cases have been reported from areas where seasonal changes are severe. This idea is now being challenged by health authorities working in tropical regions.

Heredity plays a strong role in determining the development of rheumatic fever. Wilson[6] has followed a series of over 100 families in which rheumatism was established. During a 20-year period she and her associates have charted the number of prospective cases predicated on the assumption of a hereditary influence and the number of cases which actually developed. The result strongly supported her belief in hereditary factors.

Assuming the above influences are all present in an individual there is still a requirement of unknown mechanism necessary to pro-

duce rheumatic fever. This is an atypical response, presumed to be allergic, in the body cells which creates sensitivity, but does not achieve immunity.[4] The reactions within the body which indicate that rheumatic fever may be an allergic state are briefly summarized:

1. Inflammatory changes involving the joints and the cellular contents of synovial fluid are indistinguishable from those of true allergy in which the specific antigen is known.

2. No culture from synovial fluid has yielded any bacteria or virus capable of producing these inflammatory changes in experimental animals.

3. The sulfonamides and penicillin have both failed to influence favorably the disease, although they are both capable of preventing the recurrence of acute attacks.[3]

4. Symptoms of the disease do not occur immediately following the bacterial invasion producing pharyngitis, but are delayed from three to five weeks. Swift[5] points out that just at this time a chronic focal infection may be established in the wake of an acute infection which produced an insufficient immunity.

Evidence of rheumatic disease is preceded by upper respiratory infection, particularly pharyngitis and tonsillitis, in more than 50 per cent of all cases. This association of two such common diseases of childhood[1] is the basis of our interest in this disease.

The onset of rheumatic fever may be so obscure in children that only a succession of minor events raises the suspicion of its presence. Among the earliest common symptoms are fatigue, failure to gain weight, growing pains, night sweats, recurrent low grade fever, muscular soreness, and general irritability. In such cases a history of frequent colds or tonsillitis is not unusual.

These patients may be found to have a leucocytosis and high sedimentation rate indicating that they must be carried through a prolonged period of observation. About 70 per cent of cases go undiagnosed in this phase of the disease.

More advanced stages of the disease are recognized by fever, polyarthritis, tachycardia, pallor, profuse sweating and the general appearance of a sick child. At this time in the progress of the disease the diagnosis will not be a test of one's clinical acumen.

From a small series of cases of rheumatic disease two have been selected for report, because they illustrate a serious error in manage-

ment and contrasting methods of treatment. In one the diagnosis of rheumatic fever was made and in the other it was entirely missed. From these two experiences I learned to think of rheumatic fever as potentially present in taking histories and making examinations.

REPORT OF CASES

CASE 1.—Patient E. R., male, age ten years, was seen in February 1924. He had been ill for five weeks. No physician had been called because the child did not seem seriously ill to his parents. The symptoms began with a sore throat which subsided. The child did not regain his strength, his appetite was poor, and loss of weight was noted by the parents. Night sweats had developed during the previous week. On examination there were a few prominent findings which are unusual and indicated the serious nature of his illness. These were waxy pallor, very moist skin, acetone breath, pulse rate of 110 and temperature of 100.4° F. The pharynx and tonsils showed a deep red, edematous mucosa with discrete white patches on the surface of the tonsils. The child was referred to a pediatrician who found that valvulitis was already present. He obtained a history of an older brother who had frequent attacks of polyarthritis and tonsillitis in childhood and had died at the age of 32 from heart disease.

Salicylates were given daily and the child confined to bed. At the end of three months the heart had apparently returned to normal. The tonsils and adenoids were not removed. This case was followed for three years during which time there was no recurrence of rheumatic fever.

Comment. Once rheumatic fever is established tonsillectomy should be performed only for reasons of the general health and increased resistance of the patient. Subsequent observation did not prove the operation necessary.

CASE 2. Patient S. M., female, age 13 years, was seen in April 1926. The chief complaint was frequent colds, nasal discharge and recurrent sore throat over a period of one year. The history disclosed fatigue, irritability, failure to gain weight and occasional periods of slight elevation of temperature in the afternoon. The tonsils were large, soft and contained considerable pus. The maxillary sinuses showed diminished illumination which was verified by x-ray. The family physician reported the lungs clear and the test for tuberculosis negative. Blood examination showed a normal cell count and hemoglobin of 78%. The tonsils and adenoids were removed. The

usual rise in temperature after operation persisted. On the tenth day the patient began to perspire excessively. During the third week there was epistaxis. The temperature remained slightly elevated. On the thirtieth postoperative day there were fleeting pains about the shoulders and knees. On the thirty-fourth day the child had a severe chill, a rise in temperature to 102° and precordial pain. Pericarditis with effusion followed. Rheumatic fever which had been suspected was then a certainty.

Eighteen months elapsed before this child was able to return to school and resume active life. During this period there were two attacks of pharyngitis followed by polyarthritis. There was apparent recovery, but also there was permanent cardiac damage.

Comment. Rheumatic fever should have been suspected in this case before the operation was done. The operation probably precipitated a subclinical case into a very active virulent form. At the time of operation it seemed that a marked chronic infection of the tonsils and sinuses might account for the symptoms and that improvement would follow a tonsillectomy. The occasional slight rise in temperature lasting several days and the secondary anemia had more significance than was realized.

SUMMARY

The presence of one or more minor symptoms such as fatigue, irritability, loss of appetite, low grade fever may mark the first phase of rheumatic fever. Parents bring children to the otolaryngologist frequently before consulting any other doctor, because of the presence of such symptoms. These children should be followed closely and their illnesses regarded as potential cases of rheumatic fever.

After rheumatic fever is established, the value of tonsillectomy is questionable. If these procedures are necessary for the health and increased capacity for resistance in the host they are justified. The operation should be performed in a quiescent phase of the disease when the sedimentation rate is normal and under prophylactic doses of one of the sulfonamide compounds.

The first attack of rheumatic fever changes the whole life of the victim both as to his manner of living and his life expectancy.

Approximately 50 per cent of cases are initiated by tonsillitis or pharyngitis.

Tonsillectomy before the first attack is of greatest importance.

There is a strong hereditary tendency in this disease which if present justifies the removal of apparently normal tonsils.

3245 FOURTH AVENUE.

REFERENCES

1. Cohn, A. E., and Lingg, C.: The Natural History of Rheumatic Cardiac Disease, J. A. M. A. 121:113 (Jan. 9) 1943.

2. Comroe, B. I.: Arthritis, 3rd ed., Philadelphia, Lea and Febiger, 1944, pp. 758 and 760.

3. Kuttner, A. G., and Reyersbach, G.: Prevention of Streptococcal Infections and Rheumatic Recurrences in Rheumatic Children by Prophylactic Use of Sulfanilamide, J. Clin. Investigation 22:77, 1943.

4. Lichtwitz, L.: Rheumatic Fever, New York, Grune and Stratton, 1944, p. 174.

5. Swift, Homer F.: Chapter in Cecil's Textbook of Medicine, 6th ed., Philadelphia, W. B. Saunders Co., 1945, pp. 438 and 439.

6. Wilson, M. G., et al: The Familial Epidemiology of Rheumatic Fever: Genetic and Epidemiologic Studies, J. Pediat. 22:468, 1943.

XXII

INDIVIDUAL DIFFERENCES IN HEARING FOR SPEECH

RAYMOND CARHART, Ph.D.

CAPTAIN, M.A.C., ARMY OF THE UNITED STATES

Previous reports have presented the general statistical picture of the relation, as encountered in an Aural Rehabilitation Program, between hearing acuity when measured by pure tone audiometry and when measured by speech reception techniques.[1, 2] These reports allow the conclusion that the loss for pure tones as represented by the "better ear" average for 512-2048 c.p.s. is approximately equivalent to the loss for speech reception as determined with Harvard bi-syllabic words. These reports also reveal, however, that some patients deviate markedly from the foregoing relation. It therefore is pertinent to explore the statistical and clinical aspects of individual differences between the thresholds obtained with pure tones and those obtained with speech stimuli. Various ramifications of this subject are considered in the present paper.

GENERAL PROCEDURE

The data which form the basis for this paper were taken from the files of the Acoustic Clinic at Deshon General Hospital. Except where later specified to the contrary, these data were selected at random. They all represent results of hearing tests administered to patients with impaired hearing during the routine activities of the Aural Rehabilitation Program.

The details of the testing procedure employed at Deshon General Hospital have been reported earlier.[2] Suffice it here to say that pure tone measures were made at octave intervals by the ascending threshold method. Commercial audiometers were used. The speech reception measures were made through a special amplifying system which allowed the stimulus level to be controlled through attenuation. Except where otherwise mentioned, the stimuli for testing speech reception were the Harvard bi-syllabic words (spondee lists). Speech reception stimuli were presented by live-voice.

Pure tone loss, in sensation units or decibels above normal threshold, was designated by the "better ear" average for 512-2048 c.p.s.* The speech reception score was also expressed in sensation units, this time with the normal threshold for the speech stimuli as the reference point.

Several studies were conducted to explore individual differences in acuity for speech stimuli as compared with acuity for pure tones. Information on the procedures and data for each study is most easily presented when the study is later discussed in detail. It is merely necessary at the moment to summarize the four major approaches around which the discussion which follows is organized.

The first approach was concerned with the margin of uncertainty encountered in predicting one type of threshold from the other. Predictive uncertainty was revealed by regression equations and standard errors of estimate. These statistics are summarized in the section on statistical relations.

The second approach explored the distribution, central tendencies, and variability of Difference Scores. In making this exploration, which is reported in B. of the section on statistical relations, relative acuity was separated from degree of loss by considering only the difference (for each patient) between the speech reception threshold and the 512-2048 c.p.s. average. An arbitrary convention was followed: namely, the pure tone average was subtracted from the threshold for speech reception. Thus, a negative Difference Score represented greater loss for pure tones than for speech reception. A positive score had the opposite meaning.

The third approach explored the influences of special factors on the distribution of Difference Scores. The procedure was to group patients on the basis of these special factors and then compare the Difference Scores for the various groups through Analysis of Variance and through associated small sample methods. Results are given in C. of the section on statistical relations. The special factors considered were pattern of pure tone loss, duration of hearing loss, kind of hearing loss, and acoustic trauma.

The final approach departed from statistical treatment. It was concerned with specific cases illustrating various types of patients encountered in daily routine. Case histories are summarized. The histories are representative. At the level of clinical experience, they

*"Better ear" here means that the superior threshold at each frequency was used in the average, regardless of whether at other frequencies the same ear showed the greater acuity.

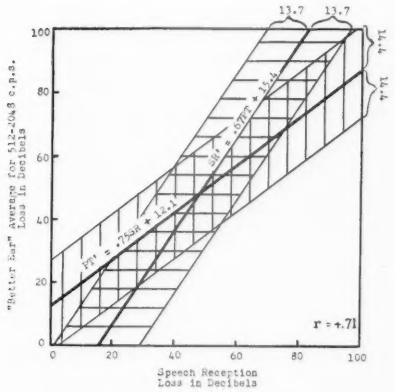


Fig. 1.—Typical regression lines illustrating the relation between speech reception threshold and "better ear" average for 512-2048 c.p.s. Coefficient of Correlation: r=+.71. Standard Error of Estimate for predicting PT'=14.4 decibels and for predicting SR'=13.7 decibels.

give insight into the reasons for individual differences in acuity for speech stimuli as compared to acuity for pure tones.

STATISTICAL RELATIONS

A. Margin of Uncertainty in Prediction of Acuity Scores. Previous studies have described in some detail both the precision with which prediction can be made from a speech reception threshold to a 512-2048 c.p.s. average and the precision with which the reverse prediction can be made.[1] Nevertheless, because the question of prediction is one aspect of the problem of individual differences, it is worthwhile to review here a typical set of results from earlier studies.

Figure 1 is illustrative of the predictive relations encountered among hard of hearing patients at Deshon General Hospital. The abscissa of Fig. 1 represents threshold for speech reception (in sensation units), while the ordinate represents average threshold for 512-2048 c.p.s. (in sensation units). The figure summarizes an analysis of the correlation between the two types of threshold as observed in a group of 100 unselected cases. (The coefficient of correlation for these 100 cases was +.71.) Two regression lines appear in the figure. One regression line (PT' = .75 SR + 12.1) represents the formula for predicting the 512-2048 c.p.s. average from the speech reception score. The other regression line (SR' = .67

PT + 15.4) represents the formula for predicting the speech reception score from the pure tone average. Each regression line is flanked by a hatched area representing the corresponding standard error of estimate. For the group under consideration, the standard error of estimate had a value of 14.4 decibels for prediction of pure tone average and 13.7 decibels for prediction of speech reception score. Fig. 1 is interpreted as follows: In predicting from a given speech reception threshold, the most likely pure tone average is the appropriate point on the PT' = SR + 12.1 line. However, measured pure tone averages showed sufficient deviation from this rule so that the best one can say is that approximately two-thirds of the scores will fall in the vertically hatched area. Therefore, applying the conventions of statistical interpretation, the margin of uncertainty in predicting any single pure tone average is 14.4 decibels in either direction from the regression line (or a total of 28.8 decibels). Similar reasoning applies to the prediction of speech reception score, where the margin of uncertainty is 13.7 decibels in either direction (or a total of 27.4 decibels). The hatched area, in other words, represents the band within which two-thirds of the predicted values may be expected to fall.

The facts presented in Fig. 1 are a sharp illustration of the repeatedly observed phenomenon that, while there is high interdependence between 512-2048 c.p.s. average and speech reception threshold, individual patients often show sharp discrepancies between the two measures of auditory acuity. Recognizing these two facts, the next question is to define further the magnitudes and incidences of discrepancies.

B. Frequency Distribution of Differences in Relative Acuity for Pure Tones and for Speech Reception. A Difference Score was computed for each patient. This Difference Score was obtained by subtracting the pure tone average from the speech reception threshold. There thus emerged for each patient a single number summarizing the discrepancy he showed between the two measures. The decibel value of this Difference Score indicated the magnitude of the discrepancy, and its algebraic sign showed the direction. A positive score indicated that the speech reception loss (in sensation units) exceeded the pure tone average. A negative score indicated the reverse.

Figure 2 illustrates the distribution of the Difference Scores for a group of 129 patients whose speech reception was measured with the Bell Telephone Intelligibility Lists. A detailed analysis of this group appears in Carhart.[1] Fig. 3 reports similar information on 1105

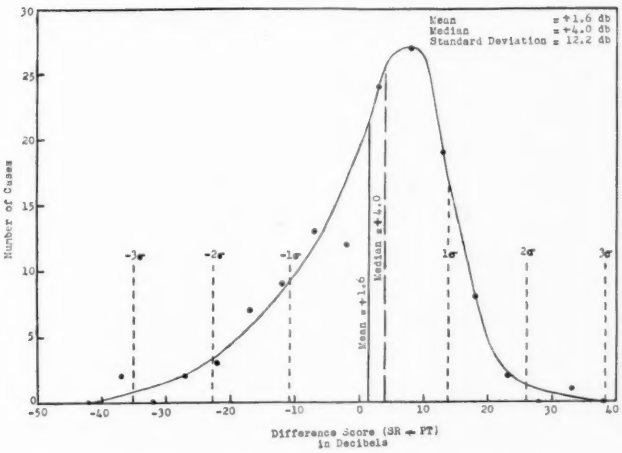


Fig. 2.—Frequency distribution of Difference Scores, derived by subtracting 512-2048 c.p.s. average from Speech Reception Score, for 129 patients whose speech reception was tested with Bell Telephone Intelligibility Lists.

patients whose speech reception was determined with the Harvard bi-syllabic word lists (spondee words). The data on these 1105 cases were selected at random from tests routinely administered from October 1944 to September 1945. They constitute an adequate sample of the entire group of tests given during this period.

It is immediately apparent that Figs. 2 and 3 illustrate highly comparable distributions of Difference Scores. The shapes of the two distribution curves do not deviate from one another to any noteworthy degree, although Fig. 2 had to be smoothed rather liberally because of irregularities due to the relatively small sample under consideration. There is also a slight displacement of the mean in Fig. 2 from that observed with the larger group. Both this displacement and a slight discrepancy between the standard deviations of the two groups can be accounted for on the basis of sample size. In general, therefore, the distribution of Difference Scores was the same when speech reception was tested with sentences as when it was tested with spondee words. The main features of the two figures may therefore be discussed simultaneously.

Figures 2 and 3 show the following characteristics which are worthy of special note.

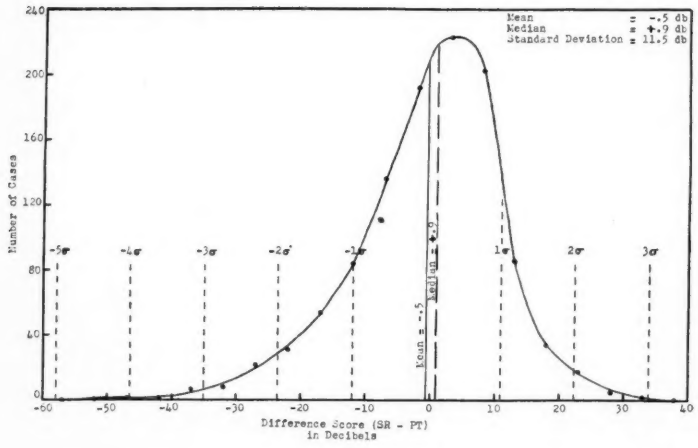


Fig. 3.—Frequency distribution of Difference Scores, derived by subtracting 512-2048 c.p.s. average from Speech Reception Score, for 1105 patients whose speech reception was tested with Harvard Bi-syllabic Words (spondees.).

1. The range of Difference Scores was very large. It extended from −52 to +33 decibels. Remembering that a score of 0 decibels means the speech reception score equalled the 512-2048 c.p.s average, it is apparent that some patients showed discrepancies which were tremendous. Later in this paper some of the reasons for such discrepancies are discussed.

2. The mean of Difference Scores was very close to zero. In other words, considering each group as a whole, the 512-2048 c.p.s average equalled the loss for speech reception (when both are computed in sensation units). The correspondence was particularly close for the group of 1105 patients, where the mean of the Difference Scores was only -.5 db.

3. In both experimental groups the median Difference Score was slightly more positive than the mean and was greater than zero. In fact, approximately 54 per cent of the area under the curve in Fig. 3 is on the positive side of zero. In other words, the loss for speech reception was greater than the pure tone average a little more than half the time.

4. The curves in Figs. 2 and 3 reveal that the differences which occurred most frequently were those immediately to the right of

the zero axis. In other words, the most common relationship was one in which the pure tone loss was slightly less than the loss for speech reception.

5. The two curves show definite negative skewing. This means that, although marked discrepancies occurred in either direction, the cases showing the greatest differences were a few having much better hearing for speech than for pure tones.

The following general conclusions emerge from the foregoing data:

While the great majority of patients showed thresholds for speech which were approximately equal to their "better ear" averages for 512-2048 c.p.s., sharp individual differences occurred and a sizable proportion of the cases evidenced discrepancies of noteworthy magnitude. Furthermore, although a slight majority of all the cases had better pure tone averages than speech reception thresholds, extreme discrepancies tended to be a little more frequent and marked for the reversed relationship.

An anticipatory word of interpretation is in order. The description given above presents an over-all picture of the distribution of discrepancies as found in a heterogeneous population. The distribution is sufficiently wide so that the findings can not all be explained as the result of limitations in the clinical accuracy of testing procedures.* It therefore becomes a problem of prime interest to search for factors which operate in individual cases to produce discrepancies that are too large to be explained in terms of the margin of testing error.

In this connection it must be clearly understood that patients having extreme positive discrepancies constitute an entirely different problem of interpretation than patients with sharply negative Differences Scores. The patient with a large positive discrepancy can be accounted for rather easily. His hearing for pure tones is better than his threshold according to speech reception. Any factor which interferes with the integration of auditory patterns into language symbols may be responsible for the observed discrepancy. Among

*The techniques used at Deshon General Hospital have been shown to have good clinical reliability when gauged by repeat tests on normal hearers. Pure tone audiometry was discovered to be reliable to within ±5 decibels and speech reception thresholds to within ±3 decibels. Repeat tests on hard of hearing patients with losses known to be constant have revealed similar reliability. While one would expect some decrease in accuracy when many types of patients are tested, mere change in reliability is not sufficient to account for the spread of distribution evident in Figs. 2 and 3.

the factors which may be operative are: unfavorable audiometric pattern, limited language ability, inferior intelligence, neurological disturbance at higher levels, and psychogenic effects. In any event, the discrepancy is psycho-acoustically credible. By contrast, the patient with marked discrepancy in the opposite direction responds to speech stimuli at a sensation level much below his thresholds for pure tones. Here the acoustic relations are such that the pure tone thresholds can not be accepted as representing the level of organic impairment in audition. Furthermore, the act of repeating speech is more complicated than the act of recognizing that a sound is present or absent. Hence, one can rule out factors of limited language ability and inferior intelligence. Similarly, one may ordinarily dismiss organic involvement of higher neurological centers. As will be clarified later, it seems safe to assume that psychological factors and fortuitous patterns of pure tone loss account for negative Difference Scores which exceed the discrepancies explainable on the basis of test reliability.

C. Variables Affecting Relative Acuity for Speech and for Pure Tone. The task of isolating factors causing discrepancy between acuity for pure tones and for speech reception is tremendous. Many variables are involved.

Without attempting a full analysis of this problem, statistical studies were made to assess the influence which several potentially important variables might have on Difference Scores. The results of these studies are reported in the paragraphs which follow.

1. Pattern of Pure Tone Loss. A previous investigation has reported in some detail the influence of audiometric pattern on speech reception threshold.[2] Five experimental groups and one control group were assembled on the basis of distinctive audiometric curves. Difference Scores were then computed for all subjects. The relative acuity, from group to group, was assessed by subjecting the Difference Scores to Analysis of Variance. The following results emerged.

a. Over a wide range of curve shapes, audiometric contour had no influence upon Difference Scores. Homogeneous groups encompassing flat losses, losses with notches beyond 2048 c.p.s., and atypical audiometric patterns were neither distinguished from one another nor from heterogeneous groups. For all the foregoing groups (at the level of statistical abstraction) the loss for speech reception equalled the 512-2048 c.p.s. average.

b. However, cases of high tone loss showed systematic displacement of their Difference Scores in the direction of better speech reception thresholds than 512-2048 c.p.s. averages. Gradual high tone loss evidenced this trend to a minor degree and the relation reached statistical significance for marked loss cases, which were defined as having increasing loss for high tones at a slope of 15 to 20 decibels per octave. For these latter cases speech reception thresholds were (at the level of statistical abstraction) over 6 decibels better than the 512-2048 c.p.s. average.

One may conclude that, while audiometric patterns may deviate widely without appreciable effect, certain types of pattern alter the relation between acuity for pure tones and for speech reception in such manner as to contribute to the wide range of Difference Scores found in heterogeneous patient groups. More specifically, since high tone loss cases show speech reception scores which tend to be superior to their 512-2048 c.p.s. averages, such cases help to explain the occurrence of negative Difference Scores.

2. Duration of Hearing Loss. The influence of the duration of loss upon Difference Scores was investigated with a series of 159 randomly selected patients. These patients were grouped into three categories, as follows:

Group 1: hard of hearing for more than 15 years (51 cases)
Group 2: hard of hearing for from 2 to 15 years (55 cases)
Group 3: hard of hearing for less than 2 years (53 cases).

When the Difference Scores for these three groups were compared by Analysis of Variance an F of 3.71 emerged under conditions where 3.06 had five per cent significance. In other words, there is strong likelihood that the three groups were distinguished from one another in terms of relative acuity for speech and for pure tones. The details were further clarified by t tests, which gave the following results.

a. Group 1 (over 15 years) and Group 2 (2-15 years) yielded a t of 0.57, which is completely insignificant. The mean Difference Score for Group 1 was —0.3 decibels and for Group 2 it was +0.7 decibels. One may conclude that the discrepancy between the means was a result of chance factors and that no important difference was found.

b. Group 1 (over 15 years) and Group 3 (under 2 years) yielded a t of 1.91, which has approximately seven per cent significance. The mean Difference Score for Group 3

was —4.1 decibels, as contrasted to —0.3 decibels for Group 1. The *t* of 1.91 is sufficiently large to raise the presumption of real discrepancy between the groups but is not large enough to be taken as definitive evidence.

c. Group 2 (2-15 years) and Group 3 (under 2 years) yielded a *t* of 2.57, which has two per cent significance. It therefore seems justifiable to take as meaningful the discrepancy of 4.8 decibels between the means for the two groups. One may conclude that there was a systematic displacement of the Difference Scores for one group in relation to those for the other.

The foregoing results allow an interesting interpretation. As a group, patients with losses of less than two years' duration yielded speech reception thresholds which were four decibels better than the 512-2048 c.p.s. averages. By contrast, patients with losses of two or more years' duration had speech reception scores which were practically equivalent to their 512-2048 c.p.s. averages. In other words, acuity as estimated by the 512-2048 c.p.s. average tended to be somewhat inferior to acuity for speech when the hearing impairment was of recent origin. The tendency disappeared when the duration of loss was greater.

Further confirmation of the above conclusion emerged when Groups 1 (over 15 years) and 2 (2-15 years) were combined and then compared by Analysis of Variance with Group 3 (under 2 years). An *F* of 7.11 resulted under conditions where 6.81 allowed one per cent confidence. The mean Difference Scores for the composite group of "old loss" cases was +0.2 decibel, which indicates close correspondence (in the aggregate) between speech reception and pure tone average. Group 3 (under 2 years) had a mean of —4.1 decibels, which represents better hearing for speech reception than the pure tone average would suggest. One may conclude that, at the level of statistical abstraction, recent loss cases are not completely comparable (in terms of the difference between acuity for speech and for pure tones) to cases of considerable duration. Thus, duration of loss is probably one of the factors accounting for the dispersion of Difference Scores encountered in heterogeneous aggregations of patients.

3. Type of Hearing Loss. A preliminary investigation was made of the influence which the kind of hearing loss, as revealed by medical diagnosis, has upon Difference Score results. The 159 cases mentioned in the preceding section were classified into six groups

on the basis of the diagnosis given them by medical officers. These groups were:

Group A: Conductive loss (21 cases)

Group B: Mixed loss, predominantly conductive (22 cases)

Group C: Mixed loss (36 cases)

Group D: Mixed loss, predominantly perceptive (13 cases)

Group E: Perceptive loss (44 cases)

Group F: Traumatizing exposure to noise (23 cases)*

When the six groups were compared by Analysis of Variance an F of 1.72 was obtained under conditions where 2.27 has five per cent significance. Such a result would ordinarily be taken as indicating that no real differential had been discovered. However, the means of the Difference Scores for the six groups distributed in such fashion that further analysis seemed justified. Table 1 presents the means of Difference Scores. One notes two interesting features.

TABLE 1.

MEANS OF DIFFERENCE SCORES FOR SIX GROUPS SEGREGATED ON THE BASIS OF MEDICAL DIAGNOSIS.

GROUP	TYPE	NO. CASES	MEAN OF DIFFERENCE SCORES
A	Conductive	21	+2.5 Decibels
B	Mixed, Pred. Conductive	22	+0.6 Decibels
C	Mixed	36	—1.7 Decibels
D	Mixed, Pred. Perceptive	13	—1.1 Decibels
E	Perceptive	44	—1.2 Decibels
F	Blast or Acoustic Trauma	23	—5.6 Decibels
Total		159	—1.2 Decibels

*At the time of the investigation it was military practice to label under a separate heading those patients whose hearing losses were service connected and were the result of blast or of prolonged exposure to noise. The cases of this type constitute Group F.

The first feature is that Group A (conductive) and Group B (mixed, predominantly conductive) have positive means: +2.5 and +0.6 decibels, respectively. Group C (mixed), Group D (mixed, predominantly perceptive) and Group E (perceptive) have negative means which range from —1.1 to —1.7. The foregoing facts suggest a differential in relative acuity for pure tones and for speech reception which is related to the dichotomy into conductive loss and perceptive loss. However, when groups were compared by *t* tests, results were nonsignificant except between Group A (conductive) and Group C (mixed), where five per cent significance was reached. Thus, the results are inconclusive, but they point to the need of further analysis of the possibility that Difference Scores are systematically displaced in relation to whether there is conductive involvement or perceptive involvement.

The second important feature of Table 1 is that Group F (blast or acoustic trauma) stands apart sharply from all other groups. The mean of Difference Scores for Group F was —5.6 decibels. As shown by *t* tests, this mean was differentiated with five per cent significance from the means for Groups C, D and E. Furthermore, it was differentiated from the means for Groups A and B by considerably better than one per cent significance. Thus, although the sampling was small, it would seem that blast and acoustic trauma cases formed a category which was different from other kinds of hearing loss. At the level of statistical abstraction, blast and acoustic trauma cases showed a systematic trend toward slightly better hearing for speech than the 512-2048 c.p.s. average would suggest. The implications of this trend are discussed in the following section.

4. Acoustic Trauma: Blast and Prolonged Exposure. Several additional investigations were made of the Difference Score distributions encountered in groups of patients wth history of acoustic trauma.

An early study of some interest involved 38 acoustic trauma cases whose speech reception scores were obtained with the Bell Telephone Intelligibility Lists. When the 38 cases were compared by Analysis of Variance with 119 cases whose hearing loss was nontraumatic in origin, an *F* of 16.1 emerged under conditions where 6.8 has one per cent significance. The mean of Difference Scores for the "trauma" group was —7.7 decibels, while the mean for the "nontrauma" group was +2.1 decibels. Thus, the total discrepancy between the two means was 9.8 decibels. Furthermore, the magnitudes and algebraic signs of the two group means indicate that (1) the "trauma" group tended to hear the Bell Telephone Intelligibility items

considerably better than the 512-2048 c.p.s. average would lead one to expect, and (2) the "non-trauma" group scored approximately the same on the two estimates of auditory acuity.

A second study repeated the one just described. In this case, however, Harvard bi-syllabic words were used to measure speech reception. The "trauma" group included 233 cases, while the "non-trauma" group contained 200 cases. An F value of 1.84 was obtained by Analysis of Variance under conditions where 3.86 has five per cent significance. The mean of Difference Scores was —1.3 decibels for the "trauma" group. It was —0.9 decibel for the "non-trauma" group. Here the "trauma" and the "non-trauma" cases failed to show any significant differentiation in relative acuity for Harvard bi-syllabic words and for pure tones as codified by the 512-2048 c.p.s. average.

Because of the contradiction in the two studies just described, it is pertinent to ask whether the manner in which traumatization occurred is a factor of importance. To this end, a series of 296 "trauma" cases were divided into two groups: 240 with histories of a single blast or concussive trauma and 56 whose losses resulted from prolonged exposure to excessive noise. These two groups were compared by Analysis of Variance with a control group of 136 cases having hearing loss of non-traumatic origin. The F value which resulted was 3.63 under conditions where 3.02 has five per cent significance. The mean of Difference Scores was —2.4 decibels in the Blast Group, —4.9 decibels in the Prolonged Exposure Group, and —0.5 in the Non-trauma Group. When the three groups were compared by t tests, the Prolonged Exposure Group was definitely differentiated (t=2.875 where 2.750 has one per cent significance) from the Non-trauma Group. Other relationships were at the level of ten per cent significance, which can not be accepted as indicating real differentiation.

The above studies on acoustic trauma are sufficiently contradictory to need interpretation. It is the writer's opinion that factors other than acoustic trauma itself were operative to produce the results just reported and that, except as these factors enter the picture, acoustic trauma cases are not separated from non-trauma cases in terms of Difference Scores. To explain: selective factors operative at Deshon General Hospital tended to load trauma groups with cases of recent origin and with cases having high tone losses. Furthermore, "trauma" groups derived from a military population subjected to war experience tended to contain a sizable proportion of patients whose hearing losses were psychogenic. As is pointed out in the

section which follows, many cases of psychogenic hearing loss have been found to yield better thresholds for speech than they do for pure tones. The relationship here has not yet been given statistical exploration. However, it seems safe to reason that, when "trauma" groups stand apart from "non-trauma" groups, the cause may be found in a high proportion of either psychogenic cases, recent loss cases, or cases with marked high tone loss. When such concentrations occur in a "trauma" group, it would be expected to show a tendency toward speech reception which is superior to 512-2048 c.p.s. average, that is, a tendency toward negative Difference Scores.

CLINICAL CASES

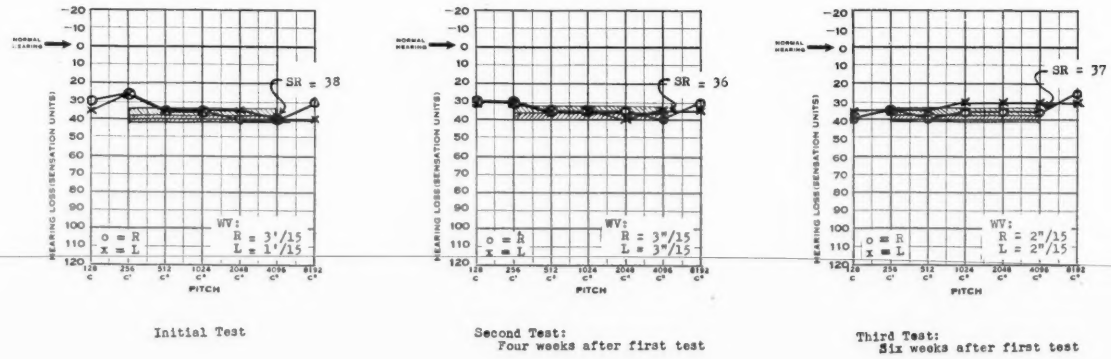
Statistical analyses, such as those reported above, give a generalized estimate of factors which contribute to individual differences in relative acuity for speech and for pure tones. Such analyses, however, are abstractions showing only trends in groups. They lose sight of the single patient and of the details which characterize his case. It is therefore pertinent to examine the problem of individual differences from the standpoint of typical cases as they are encountered in a clinical situation. Doing so sacrifices analytical precision but gives insight of an entirely different kind into the factors which influence relative acuity for pure tones and for speech. Furthermore, such a procedure highlights combinations of clinical facts and thus aids in the interpretation of new cases as they are encountered.

The following discussion presents case reports for the purpose of illustrating typical patients as observed at Deshon General Hospital. The discussion is essentially a progress report which makes no pretense of being exhaustive. However, it does emphasize factors which experience to date has shown to be significant in clinical analysis of discrepancies between acuity for pure tones and acuity for speech stimuli.

The detailed audiometric and speech reception results for each patient are shown graphically (Figs. 4-12). Pure tone thresholds are presented on the conventional audiometric charts. The concomitant speech reception thresholds are shown on the same charts, being represented by hatched bars. (For sake of clarity but without theoretical implications, each bar is 8 decibels wide and extends from 256 c.p.s. to 4096 c.p.s.) The results of standard whisper tests are also included with each set of results.

Attention may now be directed to detailed descriptions of six types of case: namely, (1) conductive hearing loss, (2) perceptive

CASE 1.—CONDUCTIVE LOSS.



Initial Test

Second Test:
Four weeks after first test

Third Test:
Six weeks after first test

Fig. 4.—Series of three hearing tests illustrating results obtained by pure tone audiometry and by speech reception for a typical case of conductive hearing loss. (SR=Speech Reception threshold; WV=Whispered Voice Test.)

hearing loss, (3) marked high tone loss, (4) psychogenic hearing loss, (5) difficulty in language perception, and (6) head injury.

A. Conductive Hearing Loss. The following case represents a typical conductive loss.

CASE 1. (1st Lt., Male, White, 28). The patient first became aware of defective hearing during a cold, which occurred about two years before admission to Deshon General Hospital. Subsequent to this cold, hearing became steadily poorer. There was no history of bleeding or discharge from the ears. The patient reported no tinnitus or vertigo. The family history was negative.

The external ears were normal. The drum membranes were intact and not remarkable. The ossicles were nonmobile. The nose, throat and eustachian orifices were normal. Bone conduction for 512 c.p.s. was prolonged. The Rinne test was negative for both ears, and the Weber test was not lateralized. The galvanic test revealed normal labyrinthine function. The general physical examination was negative, as was the neurological examination.

Three series of hearing tests were given. Each series included both speech reception and pure tone measurements. The results are reported in Fig. 4. Two facts stand out on examination of Fig. 4. First, there was high correspondence between pure tone thresholds and speech reception scores. Second, there was good agreement from one test series to another.

Case 1 is illustrative of patients who yield consistent test results characterized by Difference Scores of approximately zero decibels.

B. Perceptive Hearing Loss. The next patient represents a typical perceptive loss.

CASE 2. (Pfc., Male, White, 23). The patient had scarlet fever in 1928. His mother noted his hearing defect shortly after his recovery from this disease. There was no discharge or bleeding from the ears. The patient believes his hearing subsequently became poorer. He was not subjected to any heavy gunfire and nothing in his Army experiences seemed to make his hearing worse. The patient reported intermittent tinnitus, some paracusis, but no vertigo. His mother was hard of hearing.

The external ears were normal. The eardrums were intact but thin. The ossicles were mobile. The nose, throat and eustachian orifices were normal. Bone conduction for 512 c.p.s was absent, the Rinne test was positive bilaterally, and the Weber test was not heard. Galvanic stimulation revealed normal labyrinthine function. The general physical examination was negative, as was the neurological examination.

Three series of hearing tests, each incorporating both pure tone and speech reception measurements, are reported in Fig. 5. The test results are characterized by their consistency from one series to another and by the close correspondence between speech reception threshold and loss in the 512-2048 c.p.s. frequency band.

C. High Tone Loss. It is generally believed that, because of the acoustic nature of speech, cases with good hearing for low tones and poor hearing for high tones constitute a special category. Cer-

CASE 2.—PERCEPTIVE LOSS.

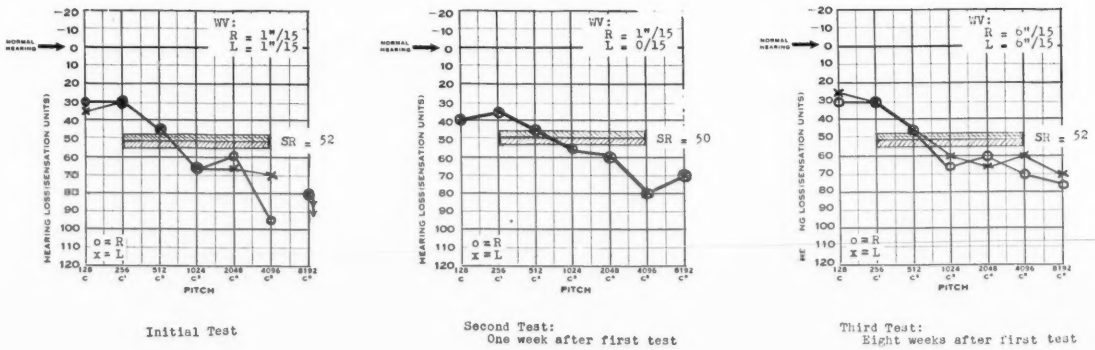


Fig. 5.—Series of three hearing tests illustrating results obtained by pure tone audiometry and by speech reception for a typical case of perceptive hearing loss. (SR=Speech Reception Threshold; WV=Whispered Voice Test.)

tainly such cases experience unique difficulties in hearing. They hear some types of sound very well, but in situations where auditory discrimination must be excellent they often experience considerable trouble. However, as is revealed by the following case, patients with marked high tone loss often have relatively stable speech reception scores. Furthermore, their speech reception scores are often quite good in comparison to their 512-2048 c.p.s. averages. (The latter fact was pointed out by the statistical analysis reported earlier.)

CASE 3. (Capt., Male, White, 39). The patient first noticed defective hearing 15 years before admission to Deshon General Hospital. The hearing became progressively worse with the passage of time. There was no history of bleeding or discharge from the ears. The patient complained of occasional tinnitus, which was once aggravated by prostigmin. There have been occasional attacks of vertigo. The patient's father, one paternal aunt, one brother, and a cousin had impaired hearing.

Inspection of the ears, nose, throat and eustachian orifices revealed no abnormalities. The ossicles, however, appeared immobile. Bone conduction was slightly diminished for 512 c.p.s. The Rinne test was negative for both ears, and the Weber test lateralized to the right. Response to galvanic stimulation revealed normal labyrinthine function. The general physical examination was negative, as was the neurological examination.

Three series of hearing tests revealed the results presented in Fig. 6. Not only does the figure reveal that there was stability from one test to another, but the speech reception threshold was consistently better than the 512-2048 c.p.s average. The Difference Scores for the three test series were, respectively, —9 decibels, —7 decibels and —10 decibels.

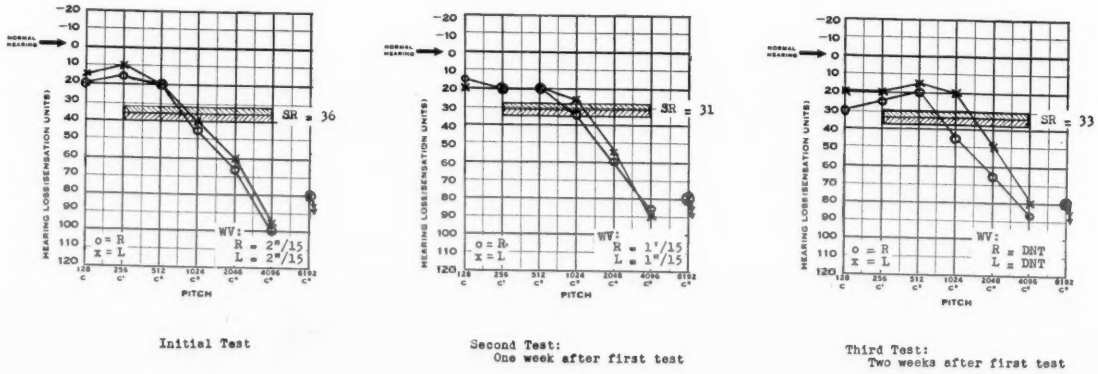
D. Psychogenic Hearing Loss. Psychogenic deafness is a clinical entity whose features have been significantly clarified by experience in Army centers for aural rehabilitation.[3] It is now clear that a sizable proportion of the patients reaching military centers for aural rehabilitation exhibit psychogenic deafness to one degree or another.* Such patients often have some organic hearing loss upon which is superimposed a "functional overlay" of non-organic origin.

Patients exhibiting either psychogenic deafness or functional overlay often reveal sharp discrepancies between pure tone measures and speech reception scores. Frequently, they also show inconsistencies from one test to another. It is dangerous to make sweeping generalizations because the manifestations can be so varied. However, three case histories will illustrate the variety encountered.

CASE 4. (T/Sgt., Male, White, 25). With the exception of several earaches without discharge in early childhood, the patient gave no history of ear trouble or

*Psychogenic deafness must be distinguished from malingering, although the borderline between the two conditions is indefinite. The important feature of psychogenic deafness is that the conscious element is sufficiently reduced so that the hearing loss, though functional, is real to the patient. Frequently, of course, the conscious element is absent.

CASE 3.—HIGH TONE LOSS.



Initial Test

Second Test:
One week after first test

Third Test:
Two weeks after first test

Fig. 6.—Series of three hearing tests illustrating results obtained by pure tone audiometry and by speech reception for a typical case of high frequency loss. (SR=Speech Reception Threshold; WV=Whispered Voice Test.)

hearing deficiency until October, 1944. At that time he was exposed during combat to artillery and mortar fire. The exposure caused marked tinnitus with impaired hearing. Hearing remained impaired. In May 1945, a mortar shell exploded near the patient. This shell wounded the patient in the back and caused hemorrhage from both ears with deafness for several hours. Hearing returned somewhat the next day but showed no further improvement prior to admission to Deshon General Hospital. The patient reported occasional tinnitus, but no vertigo. There was no history of hearing loss in the patient's family.

The external ears were normal. The tympanic membranes were intact and not remarkable. The ossicles were mobile. The nasal passages gave an adequate airway, and the eustachian orifices were unimpeded. Tuning fork tests showed that bone conduction was diminished bilaterally. The Rinne test was positive binaurally. The Weber test was not lateralized. Responses to galvanic test for vestibular reaction were normal. General physical examination was negative.

The foregoing suggests a clean-cut case of acoustic trauma. However, the audiometric results and speech reception scores were characterized by sharp discrepancies which were particularly notable since the patient reported that his hearing loss was constant rather than variable. The results of four test sequences are presented in Fig. 7. The first three were obtained within the same week. One notes that, while in the initial series the speech reception score and 512-2048 c.p.s average agree nicely, speech reception is considerably better than the pure tone thresholds in the second and third series. Furthermore, the audiometric results deviate sharply from one test to another.

On the basis of the discrepancies just mentioned a neuropsychiatric examination was requested. The neuropsychiatric diagnosis was as follows: "Conversion reaction, moderate, acute, manifested by hearing loss, in a minimally predisposed male exposed to severe military stress." Neuropsychiatric treatment was instituted and the patient's hearing was restored. The fourth test series illustrates the normal hearing evidenced by the patient following neuropsychiatric treatment.

CASE 5. (Pvt., Male, White, 27). The patient had insidious onset of defective hearing about ten years ago, cause unknown. He never had discharging ears. In June 1944, the patient suffered concussion from an 88 mm. shell which landed 25 yards away. This dazed him and caused bleeding from both ears. He had marked tinnitus and deafness for about two weeks, after which his hearing returned somewhat but never to its former acuity. There is no family history of ear trouble. At the time of admission to Deshon General Hospital the patient reported neither tinnitus, vertigo, nor paracusis.

Examination of the external ears was negative. The right drum was scarred, thickened throughout, and moderately retracted. The left drum was scarred in the anterior half, thickened and retracted. The ossicles were fixed. There was partial nasal obstruction bilaterally. The throat was negative and the eustachian orifices were patent. Tuning fork tests showed that bone conduction was diminished bilaterally. The Rinne test was positive binaurally. The Weber test was not lateralized. Labyrinthine function was normal as revealed by galvanic stimulation. The general physical examination was negative.

The audiometric and speech reception results for this patient are shown in Fig. 8. The reader will note a very startling fact: namely, that the speech reception scores were essentially normal while the loss for pure tones was at approximately the 60 decibel level. Although some instability in pure tone thresholds was evident in successive audiometric tests, the discrepancy between the two types of measure is the outstanding feature.

CASE 3.—PSYCHOGENIC HEARING LOSS.

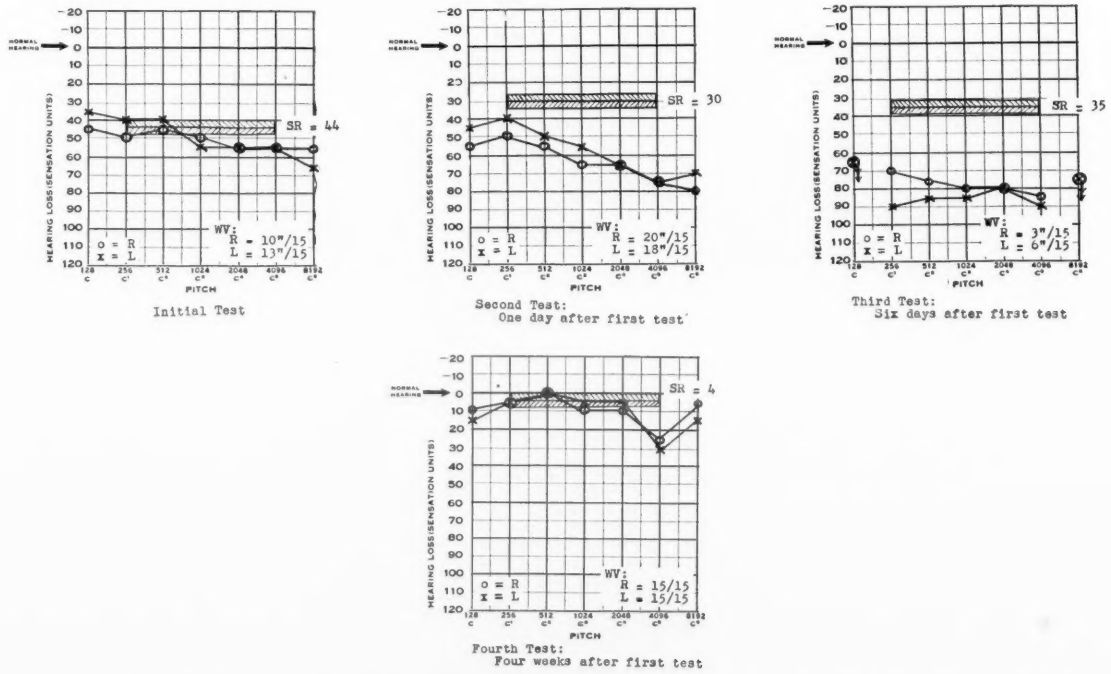


Fig. 7.—Series of four hearing tests showing results by pure tone audiometry and by speech reception for a case of psychogenic hearing loss. The first three tests preceded neuropsychiatric treatment. The fourth test illustrates recovery following neuropsychiatric treatment. (SR = Speech Reception Threshold; WV = Whispered Voice Test.)

The results just described seem paradoxical. However, a sufficient number of cases have been encountered so that the results may be accepted as authentic. Such cases, although rare, demand explanation. It seems probable that one of two conditions characterize patients with poor hearing for pure tones and good hearing for speech. They are either cases of psychogenic deafness who have undergone partial spontaneous recovery or else their psychogenic involvement has merely failed to embrace all types of sound. Cases of this type help account for the occasional Difference Score of extreme negative value.

CASE 6. (Pfc., Male, White, 22). In April 1945, the patient was riding in an armoured car which was struck by a German bazooka shell. The patient sustained severe concussion. The immediate result was transient blindness, bleeding from both ears, bilateral deafness, and marked tinnitus. Hearing in the left ear began to return in a half hour, and there was continued improvement up to the time of admission to Deshon General Hospital. Deafness in the right ear did not show comparable improvement. Except for the incident just mentioned, the history was negative.

The external canals were normal, but both drum membranes were thickened and scarred. The ossicles were mobile, the nasal airways were adequate, and the eustachian orifices were open and free. Tuning fork tests revealed that bone conduction was reduced in the left ear and absent in the right. The Rinne test was positive in the left ear but not determinable in the right. The Weber test was not heard. There was normal response to galvanic stimulation for labyrinthine function. The general physical examination was negative.

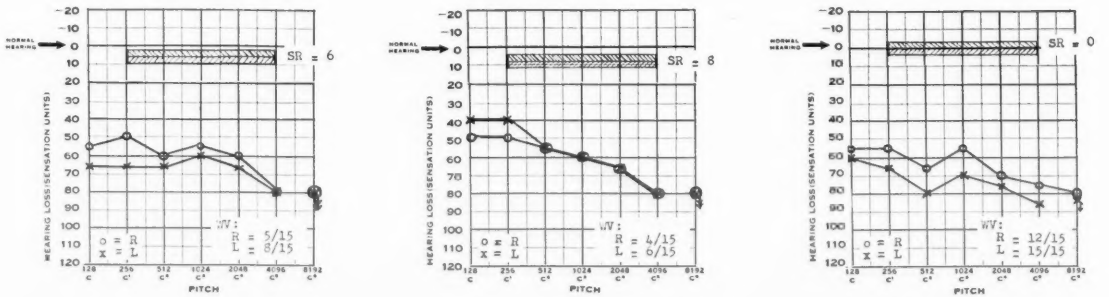
The audiometric and speech reception results are shown in Fig. 9. Comparison of the initial series and the second series reveals inconsistencies for both types of measure.

A neuropsychiatric examination yielded the following diagnosis: "Conversion reaction, acute, severe, manifested by hearing loss and transient blindness, following concussion; military stress severe; predisposition mild." Neuropsychiatric treatment was instituted. Following this treatment the third series of hearing tests was administered. One notes that there was some improvement in acuity for pure tones and a marked improvement for speech reception. (Actually, the discrepancy between the two types of measure was greater in the third series than in the initial or second test series.) The neuropsychiatrist commented on the discrepancy in the third test series as follows: "The symptomatic restoration in hearing, though complete for clinical purposes, was still marred by unconscious reservations which were evident by the failure of pure tone thresholds to show improvement comparable to that for speech reception."

E. Difficulty in Language Perception. Some patients have trouble comprehending language when spoken by others. Such patients do less well on speech reception tests than one would expect from their pure tone thresholds.

Before reporting Case 7, who typifies the patient with severe difficulty in comprehending speech, one point must be made clear. Clinical observations have shown that when presented by live-voice

CASE 5.—INCONSISTENCY BETWEEN ACUITY FOR SPEECH AND FOR PURE TONES.



Initial Test

Second Test:
Two days after first test

Third Test:
Three weeks after first test

Fig. 8.—Series of three hearing tests illustrating results for a case revealing normal hearing for speech reception and moderately severe loss for pure tones. (SR=Speech Reception Threshold; WV=Whispered Voice Test.)

the Harvard bi-syllabic word lists yielded thresholds which are highly independent of intelligence, language proficiency, and related factors. In other words, the ordinary variations in human ability do not seem to have an important effect on speech reception results, even when patients of low basic ability are among those being tested. Therefore, as the term is used here, "difficulty in language perception" means a definite abnormality in ability to understand what is being said. Even under such circumstances, a fairly definite speech reception threshold is often obtainable when the Harvard bi-syllablic words are used—although results obviously vary from one patient to another.

CASE 7. (1st Lt., Male, White, 26). According to the patient's report, hearing in the right ear was impaired for 19 years prior to admission to Deshon General Hospital. The degree of impairment remained approximately constant. For a few years prior to admission the left ear was also affected, but its acuity varied markedly and periodically. There was no history of discharging ears or of pain. The patient could not give a reason for the hearing loss, although his hearing seemed poorer during a cold or sore throat. Family history was negative.

The external ears were normal. The ear drums were both intact but were thickened. The ossicles were mobile. The nasal airway was adequate and the eustachian orifices were patent. Tuning fork tests were contradictory. Bone conduction was reduced in the left ear and no response was elicited in the right. However, the Rinne test was positive bilaterally but the Weber test was not heard. A galvanic test showed labyrinthine hypofunction on both sides. The general physical examination was negative.

The results of pure tone and speech reception tests are shown in Fig. 10. Some fluctuation of pure tone thresholds was evident, but the outstanding feature of the test results was the fact that speech reception (even for simple spondee words) was consistently poorer than the 512-2048 c.p.s. average. In everyday life, the patient had extreme difficulty understanding connected speech at levels well above the intensity at which he knew someone was talking. His difficulty in comprehension was so great that a hearing aid helped him only when he could combine lip-reading with its use.

Because of the foregoing findings a neuropsychiatric consultation was requested. After seeing the patient, the neuropsychiatrist reported central deficiency in comprehension. There was nothing to suggest organic brain disease. Periodic fluctuations in hearing indicated the possibility of a depressive substitute with the probability that the trouble was on an emotional basis.

F. Head Injury. Head injuries may lead to hearing loss. Sometimes the hearing loss is the result of damage to the inner ear or to the auditory nerve. At other times the central nervous system is affected. Finally, in many instances the head injury leaves the hearing intact. Some patients with a history of head injury have a hearing loss due to an independent cause. Thus, the involvement of auditory function differs with the site of the injury, the severity of the injury, and the extent of neurological damage. The whole picture is further complicated by the possibility that a functional over-

CASE 6.—PSYCHOGENIC HEARING LOSS.

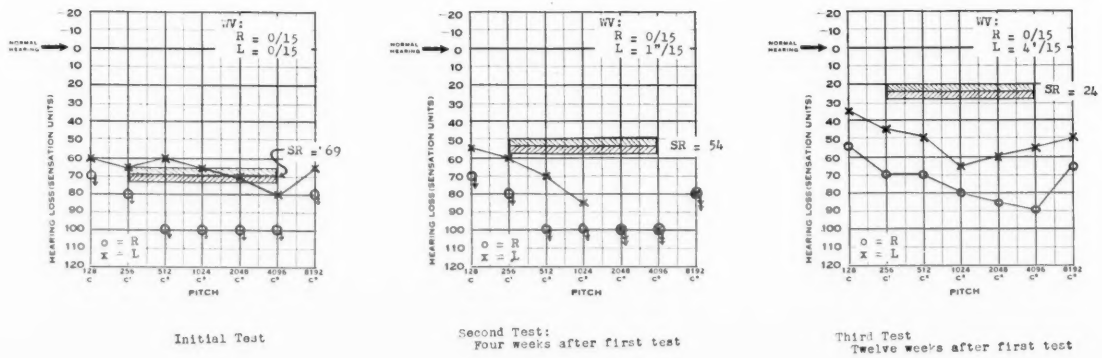


Fig. 9.—Series of three hearing tests showing results obtained by pure tone audiometry and by speech reception for a case with psychogenic hearing loss. The first two tests were administered prior to neuropsychiatric treatment. The third test shows partial restoration following treatment. (SR = Speech Reception Threshold; WV = Whispered Voice Test.)

lay may be associated with an organic loss which may be either the direct result of the injury or may be independent of it. One would expect, and one finds, that head injury cases range from patients who are completely deaf to those whose hearing remains normal. Consequently, the relation between acuity for speech and for pure tones varies widely. For these reasons, the cases presented below are illustrative but can not be called typical.

CASE 8. (Cpl., Male, White, 21). The patient entered the Army with normal hearing. In January 1945 he was struck by a recoiling 155 mm. gun. He was unconscious for 15 hours with a fractured skull and concussion. On regaining consciousness he was hard of hearing. His ears neither bled nor discharged and his hearing did not improve following the head injury. There was marked tinnitus and occasional vertigo. The patient's father was hard of hearing as a result of World War I.

The external ears were negative. The right ear drum revealed calcareous deposits but neither scars nor perforations. The left ear drum was thin and flabby. Ossicular motion was limited. The nose, throat, and eustachian orifices were negative. Tuning fork tests showed that bone conduction was reduced in the right ear and increased in the left. The Rinne test was positive for the right ear and negative for the left. The Weber test was lateralized to the left. A galvanic test gave normal labyrinthine response. The general physical examination was negative. The neurological diagnosis was: "Concussion, cerebral, severe."

Three series of hearing tests yielded the results shown in Fig. 11. The reader will observe that the three audiometric explorations were highly consistent. By contrast, the speech reception losses were, respectively, 42, 26, and 60 decibels. Such wide variation in speech reception was probably due to the effects of the cerebral concussion rather than to the high tone loss revealed by the audiograms. A provocative sidelight is the fact that the patient repeatedly reported his hearing loss to be "constant".

CASE 9. (Pvt., Male, White, 32). The patient reported his hearing loss to be the result of an automobile accident in 1940. The accident rendered him unconscious for 20 minutes. The hearing loss appeared gradually following the accident. It was not aggravated by military service. The patient experienced no tinnitus but did have occasional vertigo when admitted to Deshon General Hospital. There was no family history of hearing loss.

The external ears were negative and the drum membranes were normal. Ossicular mobility was good. The nose, throat and eustachian orifices were normal. Bone conduction was absent when tested with a 512 c.p.s. tuning fork. However, the Rinne test was negative on both sides and the Weber test was not lateralized. The galvanic test elicited normal vestibular response. The general physical examination was essentially negative. Neurological consultation was requested because the patient complained of severe frontal headaches. On neuropsychiatric examination the patient was judged, "Without psychosis or psychoneurosis, a markedly inadequate personality with defective attitude and an inefficient military record."

Three series of hearing tests yielded the results shown in Fig. 12. The three pure tone audiograms were comparable in pattern but show some variation in degree of loss. The outstanding feature, however, was the persistent superiority of speech over acuity for pure tones.

CASE 7.—DISTURBED LANGUAGE PERCEPTION.

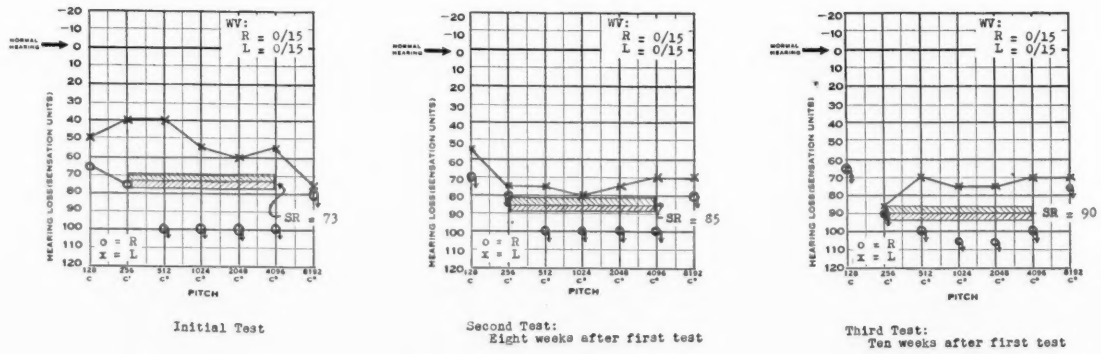


Fig. 10.—Series of three hearing tests illustrating resu'ts obtained by pure tone audiometry and by speech reception for a case having difficulty comprehending spoken language. (SR=Speech Reception Threshold; WV=Whispered Voice Test.)

It is impossible to say to what degree the hearing loss may have been the result of the head injury. However, the foregoing description includes evidence which suggests that the auditory impairment was partly independent of the head injury and partly related to it. Since functional overlay may be ruled out on the basis of the neuropsychiatric diagnosis, the case illustrates the important fact that consistently negative Difference Scores can occur when psychogenic factors are absent.

CLINICAL INTERPRETATION OF TEST RESULTS

The thoughtful person knows that, regardless of the clinical purpose for which hearing tests are used, the analysis of their implications can be made only by relating these test results to the other facts presented by the patient. Furthermore, a particular hearing test is justified only if it aids significantly in the understanding of the case. It is therefore pertinent to examine briefly the significance of adding speech reception measurement to pure tone audiometry.

Hearing tests are ordinarily given in clinical situations for either of two primary reasons. In the first place, the tests may assist the otolaryngologist in making the medical diagnosis. A noteworthy example is the combined use of air and bone conduction audiometry to check and extend the information revealed by the functional tuning fork tests. In the second place, hearing tests may assist in defining the degree and pattern of auditory impairment. They thus help the physician to assess the socio-economic implications of the patient's hearing loss. Concomitantly, they give a basis for determining what (if any) phases of aural rehabilitation will be of benefit to the patient.

It is the writer's experience that pure tone measurements plus speech reception findings add information which is of real assistance in answering certain questions that may be raised about a patient. The combined results reveal relationships which neither test can bring out when administered alone. The more important implications of the combined results may now be considered.

A. Credibility of Test Results. One great advantage of obtaining both the pure tone measurements and the speech reception threshold is the insight which is thus obtained regarding the degree of confidence with which the test results can be accepted. Close agreement between speech reception threshold and 512-2048 c.p.s. average is presumptive evidence of patient reliability. Definite disagreement should be an immediate stimulus to a careful re-evaluation of both testing procedures and of the patient. Among the reasons which may account for the disagreement between the two types of measure are the following: a pattern of pure tone loss favoring

CASE 8.--HEAD INJURY

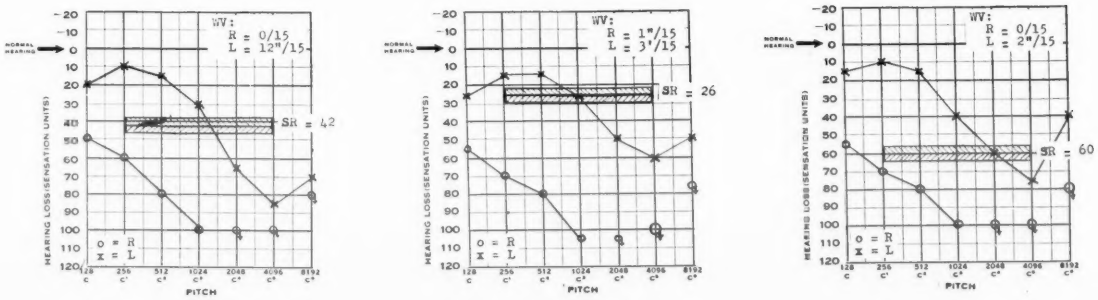


Fig. 11.—Series of three hearing tests illustrating results obtained by pure tone audiometry and by speech reception for a case with head injury. (SR=Speech Reception Threshold; WV=Whispered Voice Test.)

displacement of the speech reception score; involvement of higher neurological centers; untrustworthiness due to limited ability, to poor cooperation, or to lack of insight; deficiencies of vocabulary and perception as a result of hearing loss present at birth or acquired at an early age; insufficient mastery of English; malingering; and psychogenic involvement.

B. Variability of Hearing Loss. Repetition of combined speech reception and pure tone tests helps to assess the variability of a patient's hearing. Whenever the results for a given test series are internally consistent but there is a shift from one series to another, the presumption is raised that the patient is reliable and that the observed variation is real. Here, however, one must rule out the possibility of psychogenic involvement. Patients with a psychogenic hearing loss sometimes show rather good consistency within a test series.*

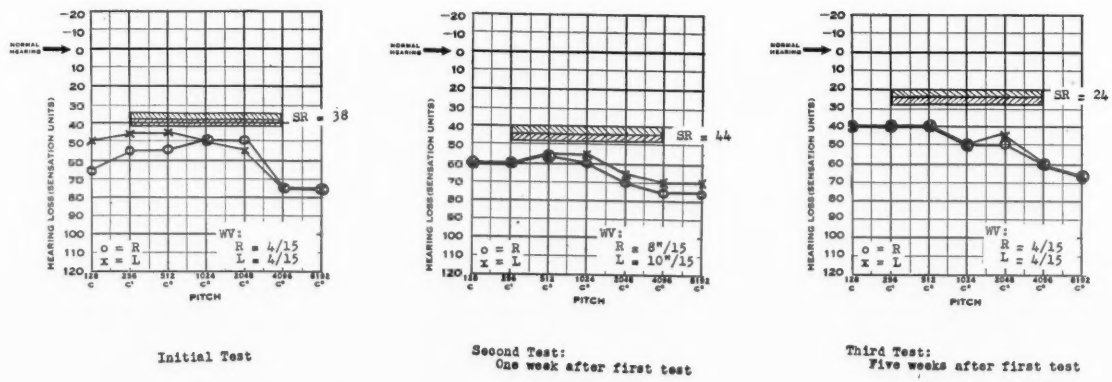
C. Systematic Differences Between Speech Reception and Pure Tone Average. When repeated test series are possible, one discovers some patients who show definite differences (between the two measures) which are nevertheless consistent from one test series to another. When such a circumstance occurs, the difference may be accepted as real and one must search for credible reasons to account for it. Among the possible reasons are those mentioned in Section A above.

D. Need for Referral. As the foregoing discussion has indicated, atypical results tend to isolate the unusual patient. When atypical results are assessed in the light of the total information on the case, one is aided in determining what referrals (if any) are indicated for the patient. Some patients will require neurological or psychiatric referrals. Others give promise of benefiting from auditory training and related educational procedures designed to counteract faulty language proficiency resulting from early loss of hearing. Still others, though atypical, will not present problems requiring referral.

E. Socio-economic Implications. A prime responsibility of the otolaryngologist is to guide the patient with permanent hearing loss in his socio-economic adjustment. A judgment must be made as to

*An independent test for functional involvement and malingering has been devised at Deshon General Hospital. The test establishes both speech reception and noise thresholds. It also measures the ability to repeat speech in background noise. Certain combinations of test results point to "non-organic hearing loss." When the results of this test are compared with independently obtained speech reception and pure tone measures, the confidence with which judgments can be made is further improved.[4]

CASE 9.—HEAD INJURY.



Initial Test

Second Test:
One week after first test

Third Test:
Five weeks after first test

Fig. 12.—Series of three hearing tests illustrating resu'ts obtained by pure tone speech reception for a case with head injury. (SR=Speech Reception Threshold; WV=Whispered Voice Test.)

the practical disadvantage which the hearing loss imposes upon the patient. Many factors must obviously be weighed in making such a judgment. The degree of loss, the pattern of loss, the prognosis, and the patient's habits of life must all be considered. It should be immediately apparent that a combination of speech reception and pure tone measures give additional insight into the subtleties of the patient's problem and thus aid in determining socio-economic implications.

Whenever the hearing loss is judged to be a definite practical handicap, the patient will need guidance in the methods which hold promise of minimizing his handicap. As already mentioned, the physician may make referrals or may recommend training programs which will be of benefit. Furthermore, if a hearing aid is indicated for the patient, he should be told what advantages he may expect from an instrument and what problems it may present for him. He should also be instructed in a practical method for procuring a good hearing aid. Once again the subtleties of the patient's problem will determine the advice he is given. Once again the extra insight gained by the combination of pure tone and speech reception findings will be useful.

SUMMARY

Individual differences in relative acuity for speech reception stimuli and for pure tones were investigated from several points of view. The purpose was to clarify the incidence and significance of these individual differences as they were observed among hard of hearing patients reaching a military program for Aural Rehabilitation. To this end, various statistical studies were made and analyses of individual case histories were employed. The test results which formed the data for this paper were obtained as part of the regular routine of the Aural Rehabilitation Program at Deshon General Hospital. Pure tone thresholds were measured with commercial audiometers. The "better ear" average for 512-2048 c.p.s. was taken as the criterion of acuity for pure tones. Speech reception thresholds were determined with the Harvard bi-syllabic words (spondees), which were presented by monitored live-voice.

The margin of uncertainty in predicting one type of threshold from the other was ascertained statistically by determining regression equations for random patient samples. While there was high interdependence between 512-2048 c.p.s. average and speech reception threshold, individual patients often showed sharp discrepancies between the two measures of auditory acuity.

The frequency distribution of Difference Scores, which were computed by subtracting 512-2048 c.p.s. average from speech reception threshold, revealed: (1) most patients showed thresholds for speech which were close to their "better ear" averages for 512-2048 c.p.s.; (2) a slight majority had better pure tone averages than speech reception thresholds; (3) extreme Difference Scores of 25 decibels or more were observed in both directions; and (4) extreme discrepancies tended to be a little more frequent and marked when acuity for speech reception was better than acuity for pure tones.

A number of statistical studies were made to assess the influence which several potentially important variables might have on Difference Scores. The basic methodology was to segregate groups in terms of the variable under consideration and subject the Difference Scores for these groups to Analysis of Variance. The following findings emerged from these studies.

1. While audiometric patterns may deviate widely without appreciable effect, certain types of pattern alter the relation between acuity for pure tones and for speech reception in such manner as to contribute to the wide range of Difference Scores found in heterogeneous groups. More specifically, high tone loss cases tend to show speech reception scores which are superior to their 512-2048 c.p.s. averages.

2. At the level of statistical abstraction, cases of recent hearing loss (under 2 years) yielded speech reception thresholds which were 4 decibels better than the 512-2048 c.p.s. averages. By contrast, patients with old hearing losses (2 years or more) showed no systematic discrepancy between the two measures of auditory acuity.

3. Difference Scores seemed independent of conductive or perceptive involvement as determined by medical diagnoses. However, this matter needs further investigation.

4. When "acoustic trauma and blast" cases were segregated from "non-trauma" cases, contradictory results were obtained. In a series of studies, significant statistical differentiation was sometimes found and was sometimes absent. The contradictory results are probably explained on the basis of selective factors which tend to load "trauma" groups with cases who for reasons other than the "trauma" itself have somewhat better acuity for speech than for pure tones.

As a statistical conclusion, several variables produce systematic shifts in Difference Score distributions. Since the present investigations make no pretense of exhausting the potentially important variables, there is need for extension of these investigations to explore the influence (if any) of other factors.

The latter portion of the paper is devoted to an analysis of case histories and to clinical interpretation of discrepancies between acuity for speech and for pure tones. Among the reasons which may account for disagreement between the two types of measure are: (1) a pattern of pure tone loss favoring displacement of the speech reception score; (2) involvement of higher neurological centers; (3) untrustworthiness of the patient due to limited ability, to poor cooperation, or to lack of insight; (4) deficiencies of vocabulary and perception as a result of hearing loss present at birth or acquired at an early age; (5) insufficient mastery of English; (6) malingering; and (7) psychogenic involvement.

The combined use of pure tone audiometry and of the speech reception technique adds important information which help one assess (1) the reliability of test results; (2) the credibility of a patient's loss; (3) the variability of his loss, (4) the referrals which may be required, and (5) the rehabilitation procedures which may be indicated.

NORTHWESTERN UNIVERSITY
EVANSTON, ILLINOIS.

REFERENCES

1. Carhart, R.: The Monitored Live-Voice As A Test of Auditory Acuity, Jour. Acous. Soc. Amer. 17:339-349, 1946.

2. Carhart, R.: Speech Reception In Relation to Pattern of Pure Tone Loss, Jour. Speech Disorders (in press).

3. Martin, N. A.: Psychogenic Deafness, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 55:81-89 (March) 1946.

4. Doerfler, L., and Stewart, K.: Malingering and Psychological Deafness, J. Speech Disorders (in press).

XXIII

FRONTAL SINUS DRAINAGE

O. E. Van Alyea, M.D.

Chicago, Ill.

The frontal sinus may become infected through extension of an infectious process from the nasal cavity or from another sinus of the anterior group, or as the result of direct introduction of virulent organisms into the sinus, such as occasionally occurs with swimming and diving.

During the onset and height of the infection, the lumen of the frontal ostium is narrowed or completely occluded by the engorged tissues which line the sinus and nasal cavities. As the swelling recedes, the ostium becomes patent and drainage is established.

Frontal sinusitis generally heals spontaneously within a week or ten days but on occasions, the infection persists until something is done to improve the drainage space. In some cases a prolongation of infection may be attributed to an organism of unusual virulence or some factor which causes a marked lowered resistance on the part of the individual. In most cases, however, the inflammation persists because of some structural defect which prevents free drainage from the sinus.

In those cases which persist despite an open ostium, drainage is impaired by one or both of two structural defects, to wit, a blocked middle meatus or encroachment on the drainage space by an ethmoid or frontal cell. It has been shown that cells either within the lumen of the sinus or encroaching on one of its walls are present at least unilaterally in 50 per cent of all individuals and that in one-third of these, the encroachment is in the neighborhood of the frontal ostium.[1] Such cells are undoubtedly potential factors in the prolongation of an inflammatory process of the frontal sinus and may well be the sole reason for the progression of an infection to a chronic state.

From the Department of Otology, Rhinology and Laryngology, University of Illinois, College of Medicine.

Presented at the Sixty-Seventh Annual Meeting of the American Laryngological Association, Chicago, Ill., May 29-30, 1946.

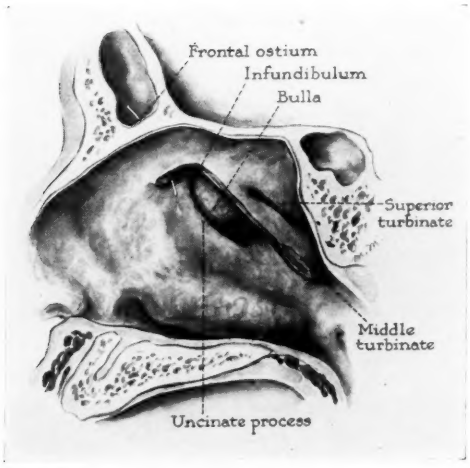


Fig. 1.—In the specimen illustrated the frontal sinus drains directly into the frontal recess. This type of drainage is found in approximately 55 per cent of cases and the ostium is usually easily reached with a cannula in cases of this type.

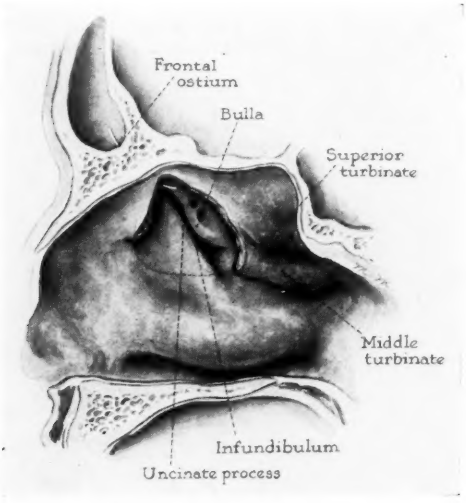


Fig. 2.—The frontal ostium in the specimen illustrated opens above the infundibulum but not into it. This type of drainage occurs in approximately 30 per cent of cases. The ostium is usually easily accessible in this type of case.

The other anatomic factor, the blocked middle meatus, in most instances is caused by an impingement of the middle turbinate against the lateral nasal wall. This may be unilateral with pressure from a deviated septum as the apparent cause. In other cases there may be no septal defect worth mentioning, yet on one or both sides the middle turbinate is in close apposition with the lateral wall. The blocked middle meatus also may be due to a cellular turbinate, the presence of polyps or may be simply the condition often observed in thin-nosed individuals in which the upper straits of the nose are unusually narrow.

With the removal of structural barriers, acute attacks of frontal sinusitis tend to subside quickly because of the ample size of the ostium and because of its location in the most dependent portion of the sinus floor. Many cases clear up immediately following infraction of the middle turbinate, the crushing of a cellular turbinate or the removal of polyps. Others tend to improve with shrinkage of the tissues of the middle meatus and in still others, displacement therapy with mild suction brings about the desired results. In those cases which do not show improvement following these procedures, irrigation of the sinus through a cannula is indicated. This may be done with comparative safety and a fair assurance of success, after the acute engorgement of the tissues has subsided. Often a single irrigation is sufficient, for in many cases a cannula which reaches the sinus penetrates or obliterates completely thin-walled cells which have been acting as barriers to normal drainage. Also, it is a recognized fact that removal of an accumulation of toxic exudate by lavage eliminates one of the causative agents of the closed ostium.

Many rhinologists hesitate to probe the frontal ostium, feeling that the trauma thus induced is harmful even though the manipulation is carried out with great care; yet it is felt that the harm caused is more than offset by the benefits attained through the establishment of improved drainage channels and the removal of exudate. Clinically this has been shown to be the case and only in isolated cases do the harmful effects of the trauma overweigh the beneficial effects of the irrigation. It should be kept in mind that the ciliated mucosa possesses great recuperative power and even though denuded of epithelium rapidly returns to normal. Many also refrain from attempting the irrigation technic believing that in order to reach the sinus the cannula tip must engage and pass through a duct. Schaeffer,[2] and other anatomic observers years ago upset the theory of the nasofrontal duct and called attention to the fact that the approach to the frontal ostium is through a comparatively wide space in most instances.

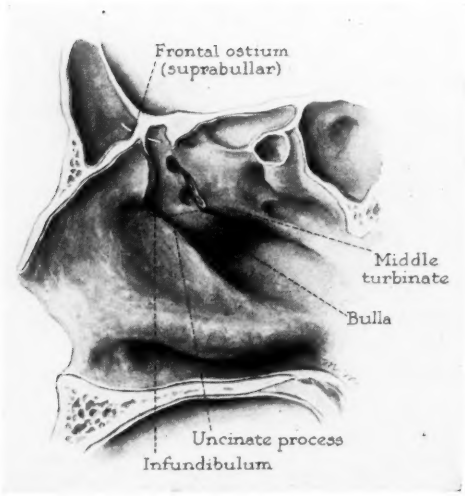


Fig. 3.—In the specimen illustrated the drainage is above the ethmoid bulla. Specimens of this type are rare occurring in approximately 1 per cent of cases.

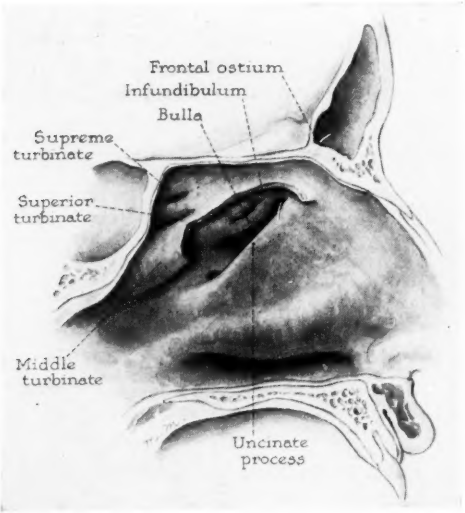


Fig. 4.—The frontal sinus is continuous with the ethmoid infundibulum. This type of drainage occurs in approximately 15 per cent of cases. To reach the sinus the cannula first must be passed into the infundibulum.

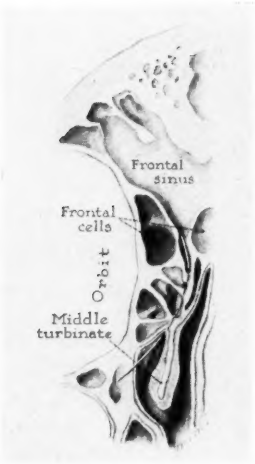
The cases which do not do well with irrigation are those of a severe toxic nature with fever and those in which local immunity has not been established. In these cases considerable swelling of the tissues is present and local manipulation accomplishes little, aside from adding to the congestion. With this type of condition, patients should be kept in bed under careful surveillance. Heat should be applied locally, anodynes given freely and proper vasoconstrictors instilled into the nose on the affected side.

As the patient becomes afebrile and shows evidence of improved resistance, local intranasal measures as outlined previously, may be considered to induce drainage. Should the symptoms, however, become more severe and show signs of intracranial or impending intracranial complications, it might be deemed advisable and imperative to establish drainage through an external opening. This may be done through a small opening in the floor of the sinus as described by Hajek,[3] Muck,[4] and Von Bajkay[5] abroad, and at various times by Crowe,[6] Lillie,[7] Boies,[8] and J. M. Brown[9] of this country. The accumulated secretions may be withdrawn by suction through the opening thus made, the sinus may be irrigated gently with warm physiologic sodium chloride solution, or merely left alone, but at no time is the ostium to be tampered with or the lining mucosa of the sinus disturbed. In conditions such as this, the sulfonamide drugs and penicillin administered internally, are of extreme value and may be the means, along with the release of toxic secretions, of effecting a cure in that which might otherwise have been a fatal case.

Recurrent Cases. A prolonged case of acute frontal sinusitis implies faulty drainage facilities with a likelihood of recurrence or progression to a chronic state. It is highly important is these cases to correct structural defects and to establish as nearly as possible adequate drainage channels.

The measures necessary for correction may be introduced during or following an acute attack. All cases should be studied from an allergic standpoint with the idea in mind that the dominant pathologic characteristic of allergy is edema and that edema blocks drainage outlets. Atypical allergy or borderline cases are constantly overlooked by the rhinologist; yet in many of these recognition and proper management remove one of the major etiologic factors in frequent nasal and sinus infections.

Structural defects such as deviated septa, impinging turbinates and polyps should be handled by proper surgical procedures. The middle turbinate may be pushed toward the septum and if cellular it



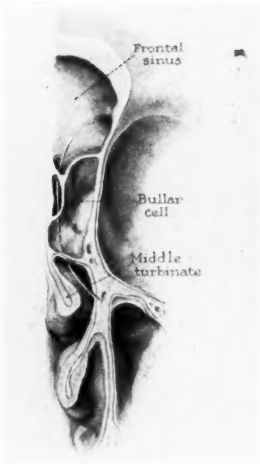


Fig. 5.—Impaired drainage due to two frontal cells which constrict the frontal ostium.

Fig. 6.—Frontal view showing unimpeded drainage from a frontal sinus.

may be crushed or the cell may be obliterated by removal of the thin lateral wall of the turbinate, but rarely is it necessary to disturb the medial or anterior surfaces.

The remaining sinuses must be carefully scrutinized as possible foci and concentrated efforts made to clear up any co-existing infection. In the examination of the sinuses, special attention is directed toward the frontal area for cells which may constrict the lumen of the frontal ostium or any other portion of its drainage pathway. The search for encroaching cells properly carried out may require diagnostic irrigations and roentgen studies with the use of iodized oil.

Cells which tend to encroach on the frontal area are often broken down by the mere passage of a cannula, and a permanent improvement in the drainage channels is established forthwith. In certain cases a cell or osseous tumor within the lumen of the frontal sinus encroaching on the ostium may be a factor in the recurring cases and responsible for the prolongation of an acute attack. These growths

require removal through the external route but this should be done with the least possible disturbance of the sinus mucosa.

Chronic Inflammatory Conditions. Most patients with long-standing suppuration of the frontal sinus may be helped immeasurably and many may be completely relieved of symptoms by the adoption of the methods outlined above for the removal of barriers to drainage.

On the other hand, in many chronic inflammatory cases radical removal of the lining sinus membranes fails to effect a cure of the disease. The removed mucosa is usually replaced by one less resistant to disease and the case often requires considerable treatment indefinitely with an occasional re-operation.

All rhinologists are familiar with the chronic nasal invalid, the multi-operated patient who haunts their offices in desperate search for relief from symptoms which are far worse than those which he had previous to his first operation.

Sewall,[10] in 1941, after observing the unfortunate results on innumerable sinus cases which had been subjected to radical procedures wrote, "Through the study of cases at revision one comes to appreciate the responsibility that is assumed by the surgeon when he removes the lining from the bony walls of any sinus."

In the radical operation, the lining membrane is completely removed on the basis of its gross appearance which is usually described as "thickened, irregular, hyperplastic mucosa." Microscopic examination, however, fails to bear out the contention of the surgeon that the membrane has undergone extensive pathologic changes and that its removal was justified. The changes which take place are those of a defensive nature with the epithelial layer relatively intact and a stroma richly endowed with powerful inflammatory cells capable of resisting invading organisms for an indefinite period of time. This membrane functions as a defense mechanism and even though it may be thickened and irregular, it is functioning to the best of its ability in a poorly draining cavity and it should be protected rather than destroyed.

Snitman,[11] in 1943, voiced his disapproval of radical procedures on the sinuses, basing his conclusions on the microscopic study of tissues removed from the sinuses of a large series of patients with chronic infections. He states, "These sinus membranes show no other changes than those of chronic inflammation. Microscopically they present every characteristic of being able to repair themselves in due

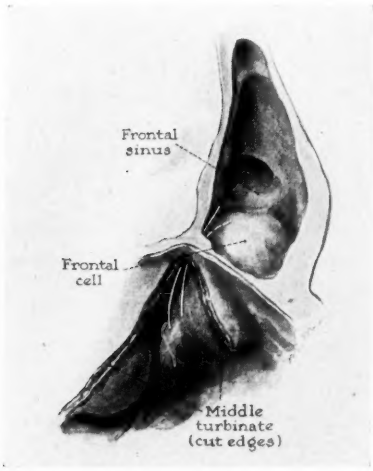


Fig. 7.—Cell in the floor of frontal sinus constricting the ostium. Frontal cells have their own separate drainage outlets in the middle meatus but in approximately 15 per cent there is an opening of communication between the cell and the frontal sinus.

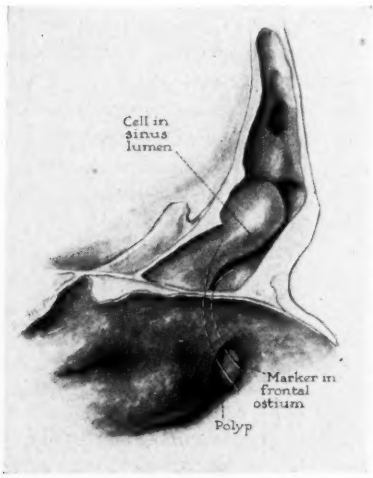


Fig. 8.—Large cell within the lumen of the frontal sinus overhanging the drainage space. A polyp in the middle meatus adds further to drainage impairment.

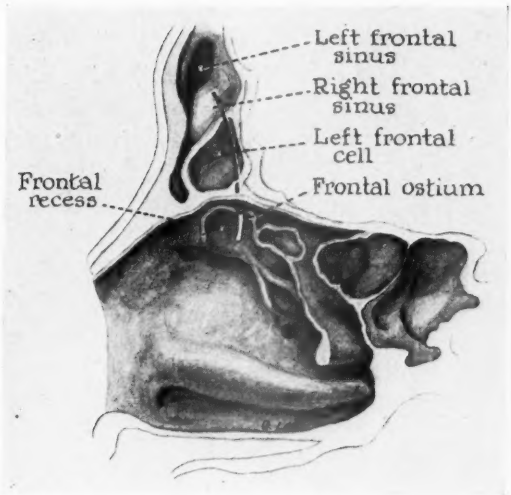


Fig. 9.—Encroachment on the terrain of the right frontal sinus by the left frontal sinus and a large cell between the two cavities.

course, when favorable conditions are provided, the most potent of which is the establishment of a functioning drainage pathway."

Certainly there will be no return of function with removal of sinus mucosa, which in resisting disease is functioning normally.

SUMMARY AND CONCLUSIONS

The health of a sinus is maintained only as long as it is lined with a functioning mucous membrane and equipped with adequate drainage facilities.

Persistence of an infection in the frontal sinus implies a complete or partial blockage of the nasofrontal passageways.

Infections, however, tend to clear up with removal of drainage barriers, and this fact clarifies the role of the rhinologist. He must direct his attention toward the restoration and maintenance of adequate drainage channels, this to be done with the least possible damage to functioning nasal and sinus structures.

During the early stages of acute frontal sinusitis, intranasal manipulations are of little value and may be harmful. With the establishment of local immunity, however, as the temperature recedes

and the acute engorgement of the tissues subsides, local measures may be introduced which will aid materially in an early termination of the disease.

Some cases respond readily to certain fairly simple procedures such as turbinal infraction, removal of polyps and sinus irrigation, but others persist and require more extensive treatment before sufficient drainage is established.

In many cases it may be necessary to correct a deviated septum in order that the middle meatus may be properly unblocked.

Co-existing infections in other sinuses should receive proper attention and every effort made to clear them up; in other cases allergy may be responsible for the maintenance of the infection and this must be properly investigated and controlled. In a final group, ethmoid and frontal cells which constrict the lumen of the frontal ostium and the nasofrontal channels must be removed.

Failure to correct frontal sinus drianage defects leads to recurrence and chronicity. The above measures, however, may be induced in the late acute stage, between attacks or during the chronic state with the assurance of considerable help, if not a complete elimination of the disease.

The alternative, the radical procedure, offers little more and with it also go the last hopes of a cure of the disease by restoration of sinus function.

The external approach to the frontal sinus is reserved for:

1. The cases in which annoying symptoms are present which have resisted all available conservative measures.

2. The release of pus under pressure in severe cases with threatening complications.

3. The removal of cells and growths within the sinus which are encroaching on the ostium.

4. The treatment of malignant growths, osteomas and inflammatory conditions with bone involvement.

135 South LaSalle St.

REFERENCES

1. Van Alyea, O. E.: Frontal Cells, Arch. Otolaryng. 34:11-23 (July) 1941.

2. Schaeffer, J. Parsons: The Nose, Paranasal Sinuses, Naso-Lacrimal Passageways and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

3. Hajek, M.: Meine Erfahringen uber die radical Behandlung der Stirnhohleneiterungen, Monatschr. f. Ohrenh. 70:641 (June) 1936.

4. Muck, O.: Heilung chronischer Stirnhohleneiterungen ohne Entstellung dursch ein chirurgisch-physikalisches Verfahren, Ztschr. f. Hals,- Nasen- u. Ohrenh. 39:477, 1936.

5. Von Bajkay, T.: Beitrage Zur Chirurgie der Stirnhohle, Monatschr. f. Ohrenh. 68:1341 (Nov.) 1934.

6. Mulligan, E. J., and Crowe, S. J.: Treatment of Acute Infection of the Frontal Sinus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:499 (June) 1941.

7. Lillie, H. I.: Personal Communication.

8. Boies, Lawrence R.: Acute Frontal Sinusitis: The Trephine Operation For Drainage in Selected Cases, Laryngoscope, vol. 52 (June) 1942.

9. Brown, J. MacKenzie: Frontal Sinusitis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:435-499 (June) 1941.

10. Sewall, E. C.: Surgery of the Nasal Sinuses, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:463-468 (June) 1941.

11. Snitman, M. F.: Interpretation of Biopsies from Sinus Operation, Eye, Ear, Nose and Throat Monthly 22:377-379 (Oct.) 1943.

XXIV

DEVELOPMENT OF THE AQUAEDUCTUS COCHLEAE AND ITS CONTAINED PERIOTIC DUCT AND COCHLEAR VEIN IN HUMAN EMBRYOS

T. H. Bast, Ph.D.

Madison, Wis.

The function and development of the aquaeductus cochleae and the periotic (perilymphatic) duct has been sparsely described. Streeter[1, 2] in his epic descriptions of the development of the periotic labyrinth briefly referred to the aquaeductus cochleae. He described it in fetuses of 85 mm. and 130 mm. where he saw it as a tubular pouch from the subarachnoid, extending toward the scala tympani, but noted no communication with the latter. Streeter writes, "As to the aquaeductus cochleae, in the 130 mm. fetus it can be plainly seen that it is already forming as a derivative of the arachnoid spaces, although the communication with the scala tympani, is not yet established. . . . The communication must be established soon after this." Streeter claimed no exhaustive study of the relationship between the scala tympani and the subarachnoid spaces and states: "a problem lies here that would be well worth careful study." Karlefors[3] noted the first anlage of the aquaeductus cochleae in 53.3 mm. and 65 mm. embryos, as a connective tissue channel between the scala tympani and the glossopharyngeal nerve. Later the connective tissue becomes loose to the extent of forming a lumen near the arachnoid. He speaks of a loose and a dense connective tissue that run parallel to each other in the aqueduct.

Recently Waltner,[4] described the region of the aqueduct in fetuses ranging from 15.8 mm. to 7 months and in a 3-day-old and a 4-week-old infant. In an 18 mm. fetus he noted the first rarefaction of the precartilage in the region of the future aqueduct. In a 27 mm. fetus he noted syncytial tissue in this area except for an island of closely packed cells which he believed later gave rise to the dura-like lining of the aquaeductus. In a 32 mm. fetus the aqueduct measured .28 mm. in diameter and the arachnoid cells surrounding the ninth cranial nerve went over directly into the syncytium of

This study is supported in part by the Central Bureau of Research of the American Otological Society.

the aqueduct. In the 37 mm. fetus he noted the cochlear vein for the first time embedded in the syncytial tissue. In a 41 mm. fetus the aqueduct both in length and width was about 0.29 mm. It was completely filled with syncytium with the cochlear vein at its center. In later fetuses the width of the aqueduct varied from one specimen to another but the average width was slightly greater. In length it increased to 2.5 mm. in a 6-month-old fetus, to 7 mm. in a 4-week-old infant. The cochlear vein he describes to lie within the syncytium of the aqueduct until the sixth month when it becomes encased by bone separating it from the aqueduct.

The above-mentioned papers present valuable information, especially on the early stages of the development of the tissue which fills the aquaeductus cochleae, with which this account agrees. There are, however, a number of structures the nature, position, and development of which are not clear. One of the reasons for this seems to lie in the terminology in general use. One is hesitant to harp on the meaning of terms long in use and yet in some cases, as in the one under consideration, a clear definition of terms is paramount. There are two terms, "aquaeductus cochleae" and "perilymphatic duct," which are commonly thought to refer to the same structure. In the above-mentioned papers only the term aquaeductus cochleae is used. I believe it was Catugno in 1760, who first discovered the bony canal extending from the bony cavity, containing the cochlea, to the cranial cavity and called it the aquaeductus cochleae, because through it fluid would escape when the stapes was pushed inward. In 1833 Breschet employed the term perilymph and endolymph. Thereafter the term perilymphatic duct was employed to refer to the channel within the bony aquaeductus cochleae which contained perilymph. The terms are generally assumed to refer to the same structure.

In 1918, Streeter suggested that the term perilymph be dropped and the term periotic fluid be substituted. Streeter also used the term periotic labyrinth to indicate the loose connective tissue with its smaller or larger fluid spaces which surround the otic (membranous) labyrinth and fill the cavity of the bony labyrinth. Such terminology clearly differentiates between the space in the bone and the connective tissue with the contained fluid which fills the bony labyrinth. In other words the term "bony labyrinth" refers to the cavity in the petrous bone when all soft structures are removed. The term "periotic (perilymphatic) labyrinth" refers to the connective tissue and its fluid spaces of mesodermal origin which largely occupy the potential space of the bony labyrinth. The term "otic (endolymphatic) labyrinth" refers to that part of the internal ear within

the periotic labyrinth derived from the otic vesicle or ectoderm. Along this same line of reasoning the term "aquaeductus cochleae" is an extension of the bony labyrinth and refers to the bony channel when all soft parts are removed. The "periotic (perilymphatic) duct" refers to the channel of loose periotic type of connective tissue and contained fluid which lies within the aquaeductus cochleae. Some such distinction between the bony channel and the contained connective tissue channel is essential to describe the embryological structures and processes which occur in the region under discussion. In the subsequent description the term "aquaeductus cochleae" will be used to indicate the channel in the otic capsule extending from the region of the round window end of the scala tympani to the cranial cavity in the region of the root of the glossopharyngeal nerve. The term "periotic duct" will be used to indicate the tube-like structure filled with periotic or arachnoid-like tissue and periotic fluid which is housed within the connective tissue that fills the aquaeductus cochleae.

The observations of the author agree with the findings of the aforementioned writers, namely, that the early precartilaginous otic capsule dedifferentiates into a mesenchymal-like tissue in the region of the future aquaeductus cochleae in a manner similar to the formation of the periotic labyrinth described by Streeter.[1] In a 25 mm. embryo the otic capsule is changing from precartilage into cartilage and the early stages of dedifferentiation of the precartilage immediately around the otic labyrinth were noted. In a 30 mm. embryo (Fig. 1) this newly formed mesenchymal tissue is clearly differentiated from the surrounding cartilaginous otic capsule. It is seen not only around the posterior ampulla (P.A.) and the cochlear duct (CO.) but also from the cochlear duct to the cranial cavity (C.C.). The connective tissue (C.T.) in this region is more reticulated toward the cranial cavity and more cellular toward the scala tympani (S.T.). The cochlear vein (V.) is seen embedded in this connective tissue. This connective tissue-filled gap of the otic capsule of the 30 mm. embryo is continuous with the connective tissue around the glossopharyngeal nerve and with the connective tissue that fills the future fossula fenestrae cochleae (niche of the round window). There is as yet no differentiation of part of this connective tissue (C.T.) into the periotic duct tissue.

In a fetus of 40 mm. (Fig. 2) the gap in the otic capsule between the cochlear duct and the cranial cavity (C.C.) is quite large and in it the looser connective tissue indicates the developing scala tympani (S.T.) and the periotic duct (P.D.) but they are not clearly

delimited from the surrounding denser connective tissue. Other structures in this area are the glossopharyngeal nerve, the cochlear vein, the fossula fenestrae cochleae and a developing bar of cartilage which gradually extends from the capsule around the posterior ampulla medially between the glossopharyngeal nerve and the periotic duct and then upward to form the floor and posterior rim of the round window. All of these structures begin to be definitely delimited at about the fetal age of eight weeks or in fetuses of 50 mm. In the subsequent development the gap in the cartilaginous otic capsule with the above-mentioned content gradually becomes subdivided until finally each of the following structures—glossopharyngeal nerve, fossula fenestrae cochleae, scala tympani, periotic duct, and cochlear vein—lies within a bony channel or compartment of the otic capsule. Accordingly there is no true aquaeductus cochleae until the bony channel, which houses the periotic duct, is formed.

In a 50 mm. fetus (Figs. 3-6) the tissue filling the areas of the scala tympani (S.T.) and the periotic duct (P.D.) is very loose and reticulated and can readily be distinguished from the surrounding denser tissue. One large fluid space is already present in the scala tympani. Around the loose reticulated tissue of the periotic duct the connective tissue is condensed to form the wall of the duct. Thus the periotic duct, already in the 50 mm. stage, has a wall of its own distinct from the surrounding connective tissue.

The cochlear vein (V.) does not lie in the reticulated tissue of the periotic duct but in the young areolar connective tissue (C.T.) outside of it. The periotic duct takes a fairly straight course between the scala tympani and the arachnoid. The cochlear vein lies medial to the periotic duct near the scala tympani but in its course toward the inferior petrosal sinus, into which it opens, it lies more ventrally and anteriorly.

About this time (50 mm. stage) a bar or shelf of cartilage sprouts out from the capsule around the posterior canal ampulla and grows medially between the glossopharyngeal nerve and the periotic duct. In the 84 mm. fetus (Fig. 8) this plate of cartilage (C.) has separated the area of the duct from the nerve. This cartilage then grows upward toward the base of the round window, the floor (C.—Fig. 11) and posterior rim (C.—Figs. 9 and 16) of which it forms. The position and extent of this cartilage bar is best seen at C. in Figs. 21, 22 and 23. This bar of cartilage is a late addition to the otic capsule. The growth of this cartilage divides the original gap in the otic capsule into three regions. One of these contains the glossopharyngeal nerve, another the fossula fenestrae cochleae (F.F.C.—

Figs. 9 and 18) and the last the periotic duct, the cochlear vein and the adjacent connective tissue. This latter area might be spoken of as the primitive aquaeductus cochleae. The rim of the round window, formed by the bar of cartilage referred to above, ossifies later than the rest of the otic capsule. It is still cartilaginous in fetuses of 230 mm. or at the age of 26 weeks. The relation of this late ossification to certain pathological processes in the round window region requires further study.

In the 100 mm. and 126 mm. fetuses (Figs. 9-12) the primitive aquaeductus cochleae is largely separated from the fossula fenestrae cochleae by the cartilage bar (C.). The periotic duct (P.D.) and the cochlear vein (V.) lie in the areolar connective tissue which fills the primitive aquaeductus cochleae. The condensed connective tissue wall of the periotic duct is clearly shown in Fig. 12. In the region where the periotic duct nears or opens into the scala tympani (Figs. 9 and 11), the vein (V.) lies medial to it, whereas near the cranial end of the duct (Figs. 10 and 11) the vein lies anterior to it. The relationship of the periotic duct to the cochlear vein is shown in Figs. 22 and 23.

At the age of 20 weeks (Figs. 13-15) the connective tissue of the primitive aquaeductus cochleae begins to be transformed into bone. In Figs. 13-15 it will be noted that the original otic capsule is largely transformed into bone and that perichondrial bone is forming. Spicules of this perichondrial bone (P.B.—Fig. 14) are invading the connective tissue between the periotic duct and the cochlear vein. At the age of 21 weeks (Figs. 16-18) this process has transformed most of the connective tissue into bone, and the cochlear vein and the periotic duct now lie within separate bone channels. That bony channel which contains the periotic duct can now be spoken of as the true aquaeductus cochleae. The periosteum of this bony aqueduct now lies adjacent to the fibrous wall of the periotic duct. These two fibrous coats have usually been referred to as the periosteum. It must be remembered, however, that the fibrous wall of the periotic duct existed long before the true periosteum developed.

Failure to distinguish in young fetuses between the periotic duct, with its contained loose periotic or arachnoid-like tissue, and the young areolar connective tissue which surrounds it has led to misinterpretation of histological sections. Thus, for example, Waltner[4] speaks of all the tissue which fills the area, which in this paper has been referred to as the primitive aquaeductus cochleae, as syncytium, except that condensation next to the capsule which he calls

dura-periosteum of the aqueduct. He says the cochlear vein lies within the syncytium but later it comes to lie within a separate bony channel. He speaks of no separate looser tissue channel which is here described as the periotic duct. In Waltner's Fig. 4, the opening of the periotic duct into the scala tympani is clearly shown. The condensed connective tissue wall of the duct is also clear, but he named this wall the round window membrane. He does not name the loose arachnoid-like tissue which fills the duct. The young areolar connective tissue which fills the rest of the gap in the otic capsule he calls the syncytium. In it lies the cochlear vein. It is evident that this section is cut to one side of the periotic duct except for its opening into the scala tympani. The tissue which he calls syncytium is the connective tissue which at this embryonic age surrounds both the periotic duct and the vein but which later will be largely replaced by bone to form two bony channels—one for the vein and one for the periotic duct.

The Periotic Duct. The early anlage for the periotic duct has already been described in the 25 mm., 30 mm. and 40 mm. fetuses and pictured in Figs. 1 and 2. In Fig. 2, the entire gap in the otic capsule is filled with a loose reticulated tissue and there is no indication of a separate duct. This condition is similar in a 62 mm. fetus (Fig. 7) although the tissue is somewhat denser next to the surrounding cartilage. In this fetus the differentiation is less advanced than in a 50 mm. fetus shown in Figs. 3-6. In the 50 mm. fetus the periotic duct is clearly differentiated from the surrounding connective tissue (Figs. 3-6). The duct is not an open tube but is filled with a reticulated tissue which is like the periotic tissue of the scala tympani or like the arachnoid, as is shown in Fig. 6. This duct tissue is surrounded by a rim of markedly condensed tissue which clearly marks off the duct from the surrounding young areolar tissue in which lies the cochlear vein. In an 84 mm. fetus (Fig. 8) the degree of development is a little more advanced than in the 50 mm.

After the wall of the periotic duct is once formed—that is, after the 50 to 84 mm. stage—the loose arachnoid content is maintained. The meshes of the reticulated tissue may become a little larger, but throughout fetal life and into the first few years of infancy this tissue occupies the potential lumen of the duct. The character of this tissue is shown in Figs. 19 and 20. In the 126 mm. fetus (Fig. 19) the meshes are small. Numerous blood cells occur in this duct tissue. In the 167 mm. fetus (Fig. 20) the tissue of the duct is looser. This looseness is partly due to the older age but partly because this fetus has a duct of large diameter. As a rule the tissue

of the duct shown in Fig. 20 is characteristic of most older fetuses. In Fig. 19, a cluster of closely packed cells is seen within the duct at C.C. A similar group of cells is seen in the duct in Figs. 4-6 and in Fig. 9. These groups of cells seen in sections represent a cord of cells which parallel the reticulated tissue of the duct. Previous authors have referred to them. The significance of these cells is not clear.

The wall of the periotic duct is best shown in Figs. 5, 12, 14, 19, and 20. It develops as a condensation of the connective tissue around the reticulated tissue of the duct. In Figs. 19 and 20 (P.D.W.) this wall consists of closely packed cells with numerous intercellular fibers stretched in one plane. This is a typical dense capsular type of areolar tissue. It is quite different from the loose areolar connective tissue (C.T.) which surrounds the duct wall.

The periotic duct is a slightly funnel-shaped tube-like channel. Its diameter is greatest at its cranial end. It narrows gradually in its course toward the scala tympani but widens again shortly before it opens into the scala tympani.

TABLE 1.—SIZE OF THE PERIOTIC DUCT IN HUMAN FETUSES. THE DIAMETER IS MEASURED AT THE LEVEL WHERE THE DUCT HAS THE SMALLEST CALIBRE.

Embryo No.	C.R. size in mm.	Age in weeks	Length of duct in mm.	Smallest Diameter of duct in mm.
E-86L	50	10+	.85	.1
E-160R	62	11+	.875	.2
E-162L	84	13+	1.25	.16
E-22L	100	15+	2.	.15
E-11L	126	16½	2.5	.2
E-105L	167	20	3.5	.25
E-21L	183	21+	?	.175
E-107L	270	30	?	.13
B-124	term	39+	4.5	.175

The diameter of the periotic duct remains remarkably constant throughout its entire period of development. There are individual variations but these cannot be related to developmental stages. The length of the periotic duct on the other hand increases progressively throughout the period of petrous bone development. A few examples of the size of the duct are given in Table 1. Cross sectional measurements of the duct were made on projected histological sections from the region of its smallest calibre. The length of the duct is hard to determine from sections unless the duct is cut lengthwise as it was for the embryos of 62 mm. and 84 mm. All

the other measurements of length were taken from models made from serial histological sections. The diameter of the duct in the 62 mm. fetus is not accurate since its wall was not yet clearly formed.

SUMMARY

1. The arachnoid-like tissue of the periotic duct develops at the same time and in like manner as the scala tympani and from the start is continuous between the tissue of the scala tympani and the subarachnoid.

2. The periotic duct, in fetuses and young infants, is filled with very loose arachnoid-like tissue and does not have a continuous epithelium-lined lumen.

3. The cochlear vein lies anterior and slightly medial to the periotic duct and not in it. Both the vein and the duct are surrounded by the connective tissue which fills the aquaeductus cochleae. After ossification of the capsule occurs, the vein lies in a separate bony canal from that of the aquaeductus cochleae, which houses only the periotic duct and its own periosteum.

4. The periotic duct is a slowly tapering channel. It is widest at its cranial end, narrows in its course toward the scala tympani but widens before it enters the latter.

UNIVERSITY OF WISCONSIN.

REFERENCES

1. Streeter, G. L.: The Development of the Scala Tympani, Scala Vestibuli and Periotic Cistern in the Human Embryo, Am. J. Anat. 21:320, 1917.

2. Streeter, G. L.: The Histogenesis and Growth of the Otic Capsule and Its Contained Periotic Tissue-Spaces in the Human Embryo. Contrib. to Embryology, No. 20 (Publication 227 of the Carnegie Inst. of Washington, pages 5-54) 1918.

3. Karlefors, J.: Die Entwicklung des Aquaeductus cochleae, Acta oto-laryng., Supp. 4, p. 69, 1924.

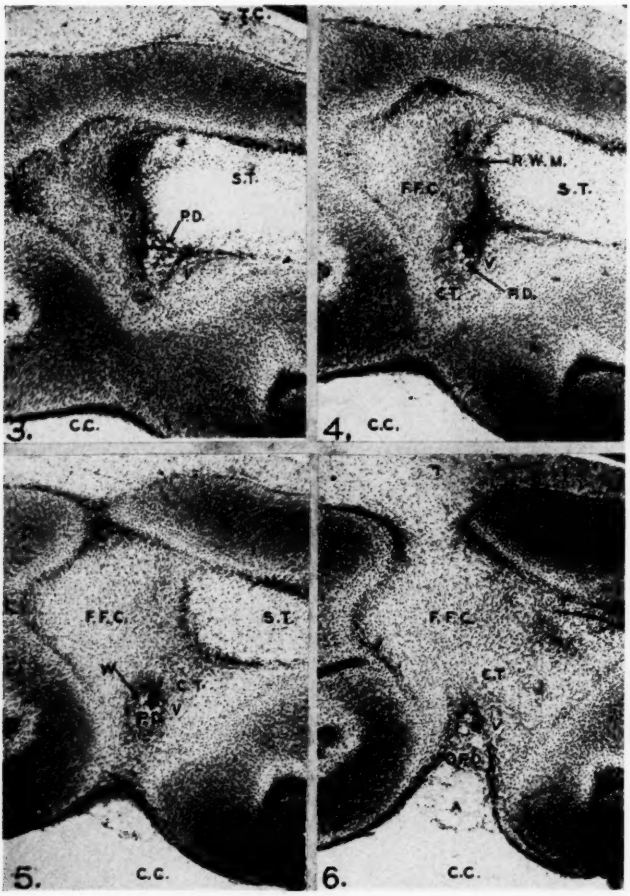
4. Waltner, J. G.: Development of the Cochlear Aqueduct and the Round Window Membrane in the Human Embryo, Arch. Otolaryng. 42:239-252 (Oct.) 1945.


T.C.
P.A.
C.C.
1.
CO.
S.V.
S.T.
F.F.C.
P.D.
C.C.
2.

Fig. 1.—Photomicrograph of a section through the otic capsule in the region of the future periotic (perilymphatic) duct of a 30 mm. (8+ week) human fetus. The area (S.T.) around the cochlear duct where the scala tympani will develop is cellular mesenchymal tissue which has dedifferentiated from precartilage. This tissue is continuous with similar tissue (C.T.) which extends to the cranial cavity in the region of the glossopharyngeal nerve (N. IX), and occupies the area of the future periotic duct, cochlear vein and fossula fenestrae cochleae. There is no cartilage between the arachnoid and the future scala tympani.

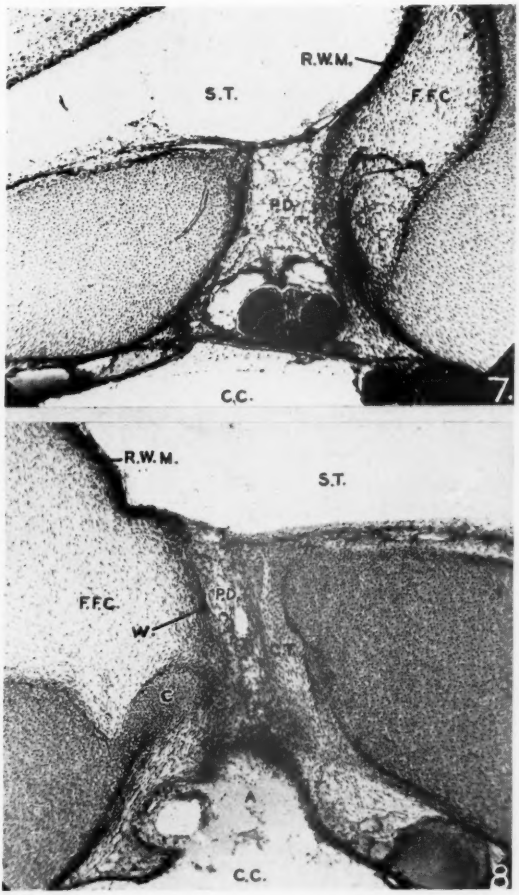
Fig. 2.—Photomicrograph of a section through the otic capsule of a 9-week-old human fetus. The connective tissue in the region of the periotic duct (P.D.), and the scala tympani (S.T.) is much looser than the rest of the connective tissue. Tissue spaces of considerable size are seen in these structures.

C.C.—Cranial cavity
C.O.—Cochlear duct
F.F.C.—Fossula fenestrae cochleae
N.IX—Glossopharyngeal nerve with surrounding ganglion
P.A.—Posterior ampulla
P.D.—Periotic duct
S.T.—Scala tympani
S.V.—Scala vestibuli
T.C.—Tympanic cavity



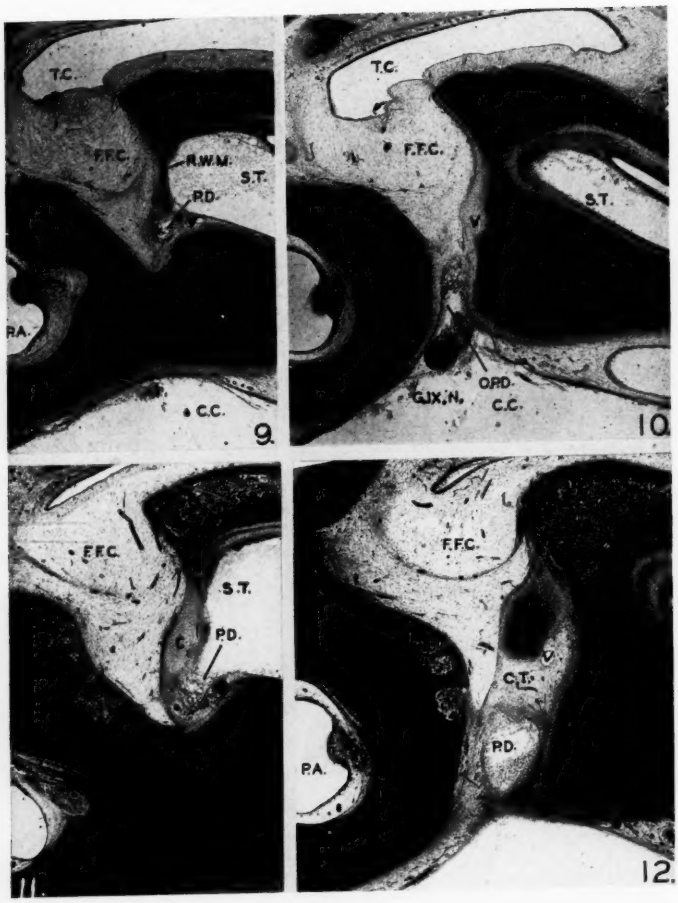
Figs. 3-6.—A series of four photomicrographs of sections through the region of the periotic (perilymphatic) duct of a 50 mm. (11 week) human fetus. These figures show the periotic duct (P.D.) from the scala tympani (S.T.—Fig. 3) to the arachnoid (A.—Fig. 6). The cochlear vein (V.) lies in the connective tissue near the periotic duct.

A.—Arachnoid
C.C.—Cranial cavity
C.T.—Young areolar connective tissue
F.F.C.—Fossula fenestrae cochleae
O.P.D.—Opening of the periotic duct into the arachnoid
P.A.—Posterior ampulla
P.D.—Periotic duct
R.W.M.—Round window membrane
S.T.—Scala tympani
T.C.—Tympanic cavity
V.—Cochlear vein
W.—Wall of periotic duct



Figs. 7 and 8.—Photomicrographs of sections through the region of the periotic duct. Fig. 7, 62 mm. (11 week) fetus; Fig. 8, 84 mm. (12+ week) fetus.

A.—Arachnoid
C.—Growing cartilage which grows upward to the round window where it will form the floor and posterior wall of said window.
C.C.—Cranial cavity
C.T.—Young areolar connective tissue
F.F.C.—Fossula fenestrae cochleae
P.D.—Periotic duct
R.W.M.—Round window membrane
S.T.—Scala tympani
W.—Wall of periotic duct

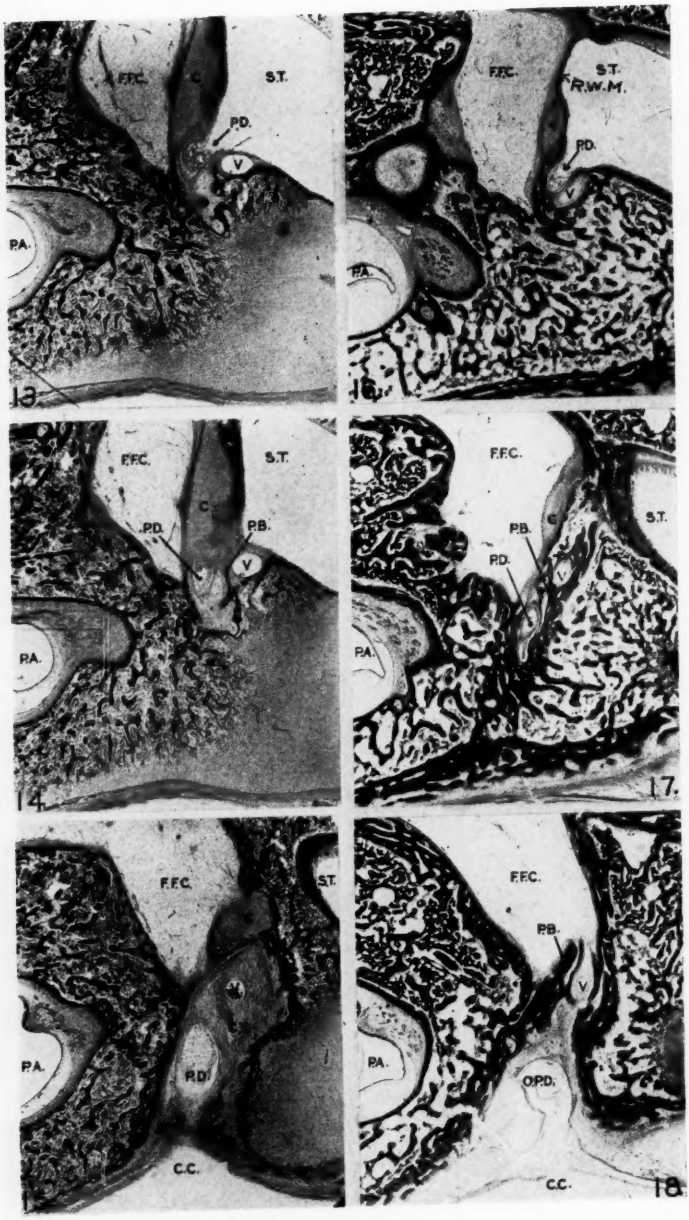

T.C.
F.F.C.
R.W.M.
S.T.
P.D.
P.A.
C.C.
9.
T.C.
F.F.C.
S.T.
G.IX.N.
O.P.D.
C.C.
10
F.F.C.
S.T.
P.D.
11.
F.F.C.
C.T.
P.D.
P.A.
12.

Figs. 9, 10, 11 and 12.—Photomicrographs of sections through the region of the round window and periotic (perilymphatic) duct.

Figs. 9 and 10, 100 mm. (14 week) fetus. In Fig. 9 the lower margin of the round window (R.W.M.) and the entrance of the periotic duct (P.D.) into the scala tympani (S.T.) are shown. The condensed connective tissue between the fossula fenestrae cochleae (F.F.C.) and the periotic duct (P.D.) becomes chondrified at a little later stage as shown in Figs. 11 to 17. The ventral and posterior part of the round window membrane are inserted into this forming cartilage. The periotic duct (P.D.) is clearly defined from the surrounding connective tissue. In Fig. 10 the periotic duct opens into the arachnoid at (O.P.D.). The cochlear vein is partly encased in young cartilage.

Figs. 11 and 12.—126 mm. (16½ week) fetus. Fig. 11 shows the opening of the periotic duct (P.D.) into the scala tympani (S.T.). The cartilaginous rim of the round window is shown at (C.). Fig. 12 shows the periotic duct (P.D.) near its opening into the arachnoid. The cochlear vein (V.) lies in the connective tissue which also surrounds the periotic duct and which fills the primitive aquaeductus cochleae. The young cartilage at (C.) is part of the cartilage bar which extends from the capsule around the posterior ampulla (P.A.) to the round window.

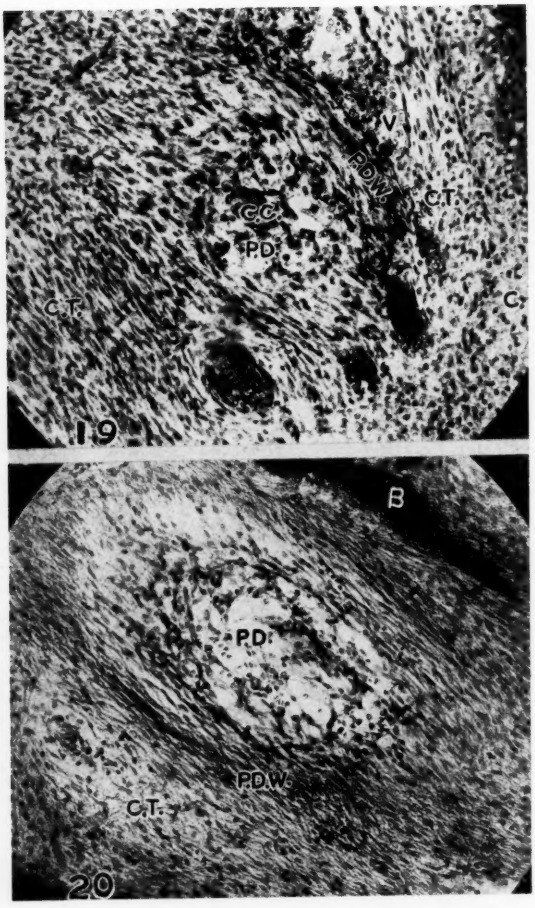
C.—Cartilage
C.C.—Cranial cavity
C.T.—Young areolar connective tissue
F.F.C.—Fossula fenestrae cochleae
G.IX.N.—Ganglion of the 9th nerve
O.P.D.—Opening of periotic duct into arachnoid
P.A.—Posterior ampulla
P.D.—Periotic duct
R.W.M.—Round window membrane
S.T.—Scala tympani
T.C.—Tympanic cavity
V.—Cochlear vein


F.F.C.
C.
S.T.
P.D.
V.
P.A.
13.
F.F.C.
S.T.
R.W.M.
P.D.
V.
P.A.
16.
F.F.C.
S.T.
C.
P.D.
P.B.
V.
P.A.
14.
F.F.C.
P.B.
C.
S.T.
P.D.
V.
P.A.
17.
F.F.C.
S.T.
V.
P.A.
P.D.
C.C.
F.F.C.
P.B.
V.
P.A.
O.P.D.
C.C.
18.

Figs. 13, 14 and 15.—Photomicrographs of sections from 167 mm. (20 week) fetus. The periotic duct (P.D.) and the cochlear vein (V.) are seen to lie within the connective tissue which fills the primitive aquaeductus cochleae. Perichondrial bone is invading the periphery of this connective tissue. In Fig. 14 a bar of membrane bone (P.B.) is separating the vein from the periotic duct.

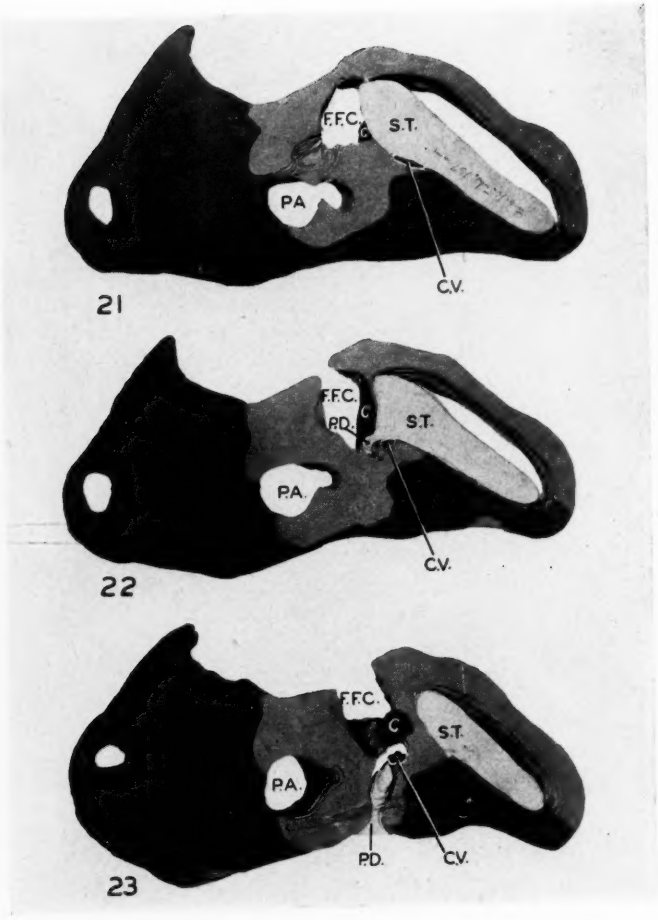
Figs. 16, 17 and 18.—Photomicrographs of sections from a 183 mm. (21 week) fetus. In these figures this new bone (P.B.) has encased the vein and also the periotic duct. The bony channel filled by the periotic duct and surrounding connective tissue is the definitive aquaeductus cochleae.

C.—Young cartilage bar which forms rim of round window.
C.C.—Cranial cavity
C.T.—Young areolar connective tissue
F.F.C.—Fossula fenestrae cochleae
O.P.D.—Opening of periotic duct into arachnoid
P.A.—Posterior ampulla
P.B.—Bar of membrane bone
P.D.—Periotic duct
R.W.M.—Round window membrane
S.T.—Scala tympani
V.—Cochlear vein


V
P.D.W.
C.T.
C.C.
P.D.
C.T.
C.
19
B
P.D.
P.D.W.
C.T.
20

Figs. 19 and 20.—Photomicrographs of sections through the periotic duct and surrounding connective tissue to show the character of the reticullated tissue within the duct and the dense fibrous tissue of the wall of the duct. Fig. 19, 126 mm. (16½ week) fetus. Fig. 20, 167 mm. (20 week) fetus.

B.—Membrane bone developing in the surrounding connective tissue
C.—Cartilaginous capsule
C.C.—Nests of cells in periotic duct tissue
C.T.—Young areolar connective tissue
P.D.—Periotic duct
P.D.W.—Wall of periotic duct
V.—Cochlear vein


F.F.C.
C
S.T.
P.A.
C.V.
21
F.F.C.
P.D.
C
S.T.
P.A.
C.V.
22
F.F.C.
C
S.T.
P.A.
P.D.
C.V.
23

Figs. 21, 22 and 23.—Photographs of three levels of a model of part of the otic capsule and part of the scala tympani, the periotic duct and the cochlear vein (167 mm., 20 week, fetus). Fig. 21 is the top part of the model, Fig. 22 is the middle and Fig. 23 the bottom. In Fig. 22 the periotic duct (P.D.) communicates with the scala tympani (S.T.). In Fig. 23 the periotic duct (P.D.) extends to the arachnoid. The position of the cochlear vein (C.V.) is shown at the three levels. The dark part of the otic capsule is cartilage, whereas the lighter part has already ossified. The bar of cartilage (C.) is a late outgrowth from the capsule around the posterior ampulla (P.A.). This cartilage grows between the glossopharyngeal nerve and the periotic duct toward the capsule around the cochlea. It forms the base of the round window (C.—Fig. 22) and then grows upward to form the posterior rim of the round window (C.—Fig. 21).

C.—Bar of cartilage
C.V.—Cochlear nerve
F.F.C.—Fossula fenestrae cochleae
P.A.—Posterior ampulla
P.D.—Periotic duct
S.T.—Scala tympani

XXV

THE INFLUENCE OF THE SULFONAMIDES AND ANTI-BIOTICS ON THE FUTURE OF OTOLARYNGOLOGY

BERNARD J. MCMAHON, M.D.

ST. LOUIS, MO.

There are many factors which may influence otolaryngologic trends over the course of years. At times these factors may be clearly apparent, while again they may be more insidious and gradual and thus difficult to evaluate.

We have seen the treatment of sinusitis change either to the radical or the conservative in a locality as a result of the influence of one who holds the respect of his fellow otolaryngologists and has thus shaped the policy, however unconsciously, of the conduct of his associates, probably by reason of his stronger medical or surgical inclinations respectively towards otolaryngologic problems. The pattern of infections, which may change from year to year or cyclically, is another important factor of which we are all fully aware. These variable endemic episodes are as frequently the result of the occurrence of different strains as of entirely different bacteria or of viruses which show variations in resistance to therapeutic agents, especially since the advent of chemotherapy. The lack of recognition of these factors may often lead to false and unwarranted modifications of therapeutic methods.

One of the earlier flurries in chemotherapy occurred in 1919 when Young and his collaborators of Baltimore,[1] introduced mercurochrome 220 as a urinary antiseptic for the gonococcus, the B. coli and the staphylococcus, and later in 1924 when Young and Hill[2] advocated it as a germicide with great promise in the treatment of septicemia caused by B. coli and staphylococcus when used intravenously. Within these years mercurochrome was used very extensively in the treatment of a variety of infections and many glowing

---

From the Department of Otolaryngology, St. Louis University School of Medicine, St. Louis, Missouri.

Read before the Sixty-Seventh Annual Meeting of the American Laryngological Association, Chicago, Illinois, May 30, 1946.

reports of its efficacy were published. However, this enthusiasm about its therapeutic value as a deep tissue germicide was short lived and its use was abandoned.

It is an accepted fact that the sulfonamides and penicillin exert a beneficial effect upon infection of the upper and lower respiratory tract. Reports of animal experimentation, and clinical results have proven conclusively that they deserve a place in our armamentarium in combatting and controlling the pathological changes induced in the body tissues by the invasion of certain micro-organisms. That other micro-organisms are invulnerable to these drugs by reason of their innate properties is apparent from the clinical fact that we are unable to influence or control the course of some diseases by the adequate administration of such drugs. We speak principally of penicillin because of its more general use. Tyrothricin and gramicidin also have a definite place in antibiotic therapy, when used locally, but in a much more limited manner. Streptomycin therapy is still in an early experimental stage.

Experimental evidence has pointed to the conclusion that the sulfonamides are bacteriostatic in action[3] and clinical experience has substantiated this conclusion. On the other hand the action of penicillin is said to be not only bacteriostatic but bactericidal as well, according to Chain and Duthie.[4]

The adverse results that have been reported have been due to the biological resistance of the micro-organism or the intolerance of the patient. The former is either a natural characteristic of certain bacteria or an acquired property as a result of small inadequate clinical doses or frequent short courses of treatment with either the sulfonamides or penicillin. Abraham, Chain and others[3] have rendered a culture of staphylococcus aureus, grown in vitro in increasing concentrations of penicillin, resistant to the drug, and Rammelkamp and Maxon[5] have similarly increased the resistance of two strains of staphylococci. This phenomenon was later corroborated by McKee and Houck with staphylococci, pneumococci and streptococci,[6] and also by Schmidt and Sealer with pneumococci.[7]

The intolerance of the patient to sulfonamides is much more frequently encountered than it is to penicillin. This intolerance is manifested by a variety of reactions from a localized wheal to a generalized erythema, or from a mucosal reaction of the nose to an alarming asthmatoid diathesis.[8] Such reactions following the administration of penicillin are less common than post-sulphonamide reactions, although they do occur, and should be recognized. Certain

patients are inherently sensitive to the molds of penicillium notatum. This was strikingly illustrated in a child who was referred by a pediatrician for the removal of a suspected nonopaque foreign body from the right main bronchus. This diagnosis was concurred in by the radiologist who reported a moderate atelectasis of the inferior half of the right lung. A bronchoscopy was performed which revealed an engorgement and edema of the right main bronchus, but no foreign body. On subsequent questioning we ascertained that the patient had been receiving penicillin for several days for an acute respiratory infection. This was withdrawn and within 48 hours all pulmonary signs and symptoms subsided.

It is obvious that chemotherapeutic agents, in order to be effective, must gain access to the foci which they are intended to reach. Therefore, an adequate local circulation is a sine qua non for the fulfillment of this purpose as well as for that of carrying the phagocytic cells to the area. The drug must be brought into contact with the vulnerable micro-organisms in sufficiently high concentration and this contact must be maintained for a sufficient length of time to enable the body defenses to elaborate the necessary phagocytes to destroy the invading organisms.

It has been repeatedly stated that the action of these drugs is bacteriostatic, but it has never been explained how organisms of attenuated virulence can contribute sufficient stimulus to the hematopoietic and the reticulo-endothelial systems to adequately and successfully activate the formation of leucocytes, histiocytes and other phagocytic and reparative cells. The explanation may rest in the fact that these drugs do not completely interrupt cellular respiration, inasmuch as bacterial cells inhibited in their growth continue to metabolize even in the presence of an excess of sulfonamides.[9] Since the leucocytes are the chief phagocytic cells which are being elaborated during bacteriostasis, a special study of the effect of the sulfonamides, penicillin and other drugs on the viability and activity of these cells was conducted by Abraham, Chain, Florey and others[3] in 1941. They reported that while leucocytic activity was retarded or completely abolished by sulfathiazole, it was not affected by penicillin solutions in similar dilutions. Dubos,[9] states that "The occurrence of secondary irreversible alterations during bacteriostasis may also explain the observation that the bacteriostatic effect of the sulfonamides can be converted into a bactericidal effect by raising the temperature at which the reaction is taking place from 37° C. to 40° C." A similar bactericidal effect of sulfanilamide was observed by Wolff and Julius[10] in 1939. Also, Hobby, Meyer and Chaffee[11] in 1942 showed

that penicillin exerted a powerful bactericidal action on streptococci, pneumococci and staphylococci when acting under conditions favorable to their growth, 99 per cent of the organisms being killed. This conclusion was confirmed by Chain and Duthie[4] in 1945.

From the foregoing statements we are therefore justified in assuming that both the sulfonamides and penicillin are bactericidal as well as bacteriostatic.

In evaluating the effectiveness of the sulfonamides and penicillin, their clinical limitations and comparative advantages should be realized fully. We are familiar with the specific bactericidal properties of these substances, the contraindications to the administration of the sulfonamides and to a lesser degree to the administration of penicillin. We know that the sulfonamides are rendered less active in the presence of necrotic tissue and exudates because of the para-aminobenzoic acid and the other substances which reverse the action of sulfonamides and which are produced by many cells and released during tissue breakdown.[9] Penicillin on the other hand does not affect the viability of leucocytes except in high concentrations and is consequently more effective under such conditions.[3]

Significant observations were made by Abraham, Chain, et al,[3] by Rammelkamp and Maxon,[5] and by McKee and Houck,[6] relative to the resistance induced by penicillin in certain strains of staphylococcus aureus and pneumococci, types I, II and III. These investigators found that repeated serial passages of these micro-organisms through cultures containing penicillin in increasing amounts resulted in a greatly increased resistance to the penicillin, while at the same time there occurred a marked decrease in the virulence of the micro-organisms for mice, though less so in the type I and II pneumococci. Furthermore, the resistance to penicillin was not decreased or the virulence increased by repeated passages of the cultures through mice. It is significant that penicillin-resistant strains are not resistant to sulfonamides and sulfonamide-resistant strains are not resistant to penicillin, unless such resistance is induced by the specific drug.[12, 13]

In discussing the use of the sulfonamides and penicillin it is timely to comment upon the manner in which they are used and applied. It is generally conceded, as we have mentioned, that these drugs must be in contact with the micro-organisms in sufficiently high concentration for a sufficient length of time to retard the growth of the organisms in order to allow the leucocytes and other phagocytes to be carried to the area of infection, destroy the bacteria and bring the infection under control. The tissue sulfonamides and

penicillin cannot penetrate or diffuse through well-established fibrosed cavity walls since the circulation is retarded by blood vessels which are narrowed because of a thickening of their walls, or completely obliterated as a result of compression by the fibrosed tissue which they traverse. In an acute infection the neighboring lymphatics and capillaries are so completely blocked by fibrin and histiocytic cells after 24 hours that the blood and lymph streams are reversed and diverted away from or around the focus of infection.[14-16] At present the sulfonamides are administered by mouth, locally, and by lozenges and gum. Penicillin is similarly administered and in addition is given parenterally, intrathecally and by inhalation.

We feel that applications of either of these substances to the surface of a mucous membrane of the respiratory tract is entirely unavailing and futile. The pathogenic micro-organisms are in the subepithelial tissues, while the surface epithelial cells are either swollen, and relatively impermeable, or they have sloughed away. In either case the stream of transudation is to the surface of the mucosa, so that it is impossible for the fluid or substance to penetrate the subepithelial tissues in the face of the greater pressure. If the cilia are functioning, any substance imposed upon their surface is carried back to the portal of entry of the cavity or, as in the nose, to the nasopharynx, in a relatively short time. The germicidal activity of penicillin in a cavity only serves to retard the growth of or kill such bacteria as are in the cavity and thus prevent the spread of viable bacteria to other localities.

We are thus forced to conclude that intranasal or intratracheal administration of solutions containing the sulfonamides or penicillin is totally inadequate in enhancing any results accomplished by the use of these drugs when carried through tissue channels to penetrable areas of infection. Mutch and Rewell[18] state, however, that calcium penicillin administered as a mist is very rapidly absorbed through the respiratory mucous membrane, and that the concentration in the blood subsequently approximates that found after the injection by intramuscular and intravenous routes.

The fact that such solutions frequently contain ephedrine or other synthetic tissue constrictors does not in any way enhance their value, because when these drugs are in the form of emulsions, the mechanically irritating effect of the crystals will more than offset the possible shrinking effects of the vehicle, assuming that this in itself has no irritating properties.

In recent years there has been an inclination on the part of some otolaryngologists to seek more highly specialized fields within

their specialty as a result of the temporary restrictions of their activities supposedly from the use of the sulfonamides and the antibiotics.

It is true to some extent that there has been a reduced incidence of the more serious sequelae of otitis media, sinusitis, tonsillitis and deep tissue infections of the neck, but it is likewise true that there has been a much greater incidence of relapses or recurrences following these infections. Our own observations are corroborated in a report by Spink, et al,[17] who conducted a survey of 210 young men suffering from acute infections of the upper respiratory tract. They concluded that "Sulfadiazine did not shorten the clinical course of patients with tonsillitis or eradicate hemolytic streptococci from the pharynx—although it did appear to diminish the severity of the disease in severely ill patients," and also "that clinical relapses following the administration of penicillin were frequently encountered and appeared to be related to the reappearance of hemolytic streptococci in the throat."

This syndrome of diminution of symptoms and clinical relapses corroborates the experimental evidence that these drugs have principally a bacteriostatic action and that certain strains of micro-organisms must become sulfonamide-resistant or penicillin resistant, but continue their biological growth and resume their multiplying activities when the drugs are withdrawn. The practical and ominous clinical application of this phenomenon lies in its potentialities regarding the transmission in epidemic proportions, of sulfonamide-resistant micro-organisms of unattenuated virulence through a communty since such bacteria do not lose their sulfonamide-resistant qualities, but do acquire increased virulence by animal passage.

Penicillin-resistant micro-organisms likewise lose their virulence while retaining their ability to multiply on transfers in vitro and on animal passage without regaining either their sensitiveness to penicillin or their virulence.[6] This does not necessarily mean that such micro-organisms are incapable of transmitting infection, since both of the above faculties are relative and will be modified by the reactions of the individual hosts. The recurrence of these micro-organisms is, as a rule, in symbiosis with other pathogens and of equal importance under such circumstances. We are, therefore, faced with the fact that the application of the sulfonamides and the antibiotics in the control of respiratory infection is limited. It is of interest to conjecture whether in the future we shall not revert to the usual medical and surgical methods of treating respiratory infections and their complications because of the presence simultane-

ously of sulfonamide-resistant and penicillin-resistant micro-organisms, which require enormous doses of these drugs, beyond the tolerance of the patient.

Any consideration of a modification of otolaryngologic teaching and methods is untimely. We are agreed, I am sure, that there is no system of therapeutics which should not be accompanied by careful observation of the locus which the medication is intended to influence. In chemotherapy particularly there is an undeniable masking of symptoms which at times requires the most careful and accurate diagnostic acumen possessed by a well trained otolaryngologist, for proper evaluation.

The younger otolaryngologist should be even more carefully instructed and better trained. More time should be devoted to the teaching of immunology, physiology, biochemistry, endocrinology and pathology in their relations to otolaryngology. A fuller and more accurate knowledge of these subjects will make him more capable in the use of chemotherapy, as applied to the treatment of respiratory diseases and of certain types of deafness. This should not be at the expense of a thorough general surgical training especially in surgery of the head, neck and thorax during his internship, preliminary to his basic training in otolaryngology.

The present military emergency, which has interfered with proper and orderly medical studies and training, has been most keenly felt by the physician in the specialties. There has been a dehiscence which it is the duty of the educators engaged in graduate medical teaching to bridge over. There will of necessity be a period during which the ideal plan cannot be fully operative, but in time this compromise will be adequately met and the final desideratum fully achieved.

SUMMARY

1. The sulfonamides and the antibiotics have a definite place in the treatment of otolaryngologic conditions.

2. Their uses are limited by the sensitiveness of the patient to these drugs, by the acquired "sulfonamide-resistance" or "penicillin-resistance" of the micro-organisms, and by the potential dangers of masked symptoms.

634 North Grand Avenue.

REFERENCES

1. Young, H. H., White, E. C., and Swartz, E. O.: A New Germicide for Use in the Genito-Urinary Tract, "Mercurochrome 220", J. A. M. A. 73:1483 (Nov. 19) 1919.

2. Young, H. H., and Hill, J. H.: The Treatment of Septicemia and Local Infections by Intravenous Injections of Mercurochrome 220 Soluble and of Gentian Violet, J. A. M. A. 82:669 (Mar. 1) 1924.

3. Abraham, E. P., Chain, E., Florey, H. W., et al: Further Observations on Penicillin, Lancet 11:177 (Aug. 16) 1941.

4. Chain, E., and Duthie, E. S.: Bactericidal and Bacteriolytic Actions of Penicillin on the Staphylococci, Lancet 1:652 (May 26) 1945.

5. Rammelkamp, C. H., and Maxon, T.: Resistance of Staphylococcus Aureus to the Action of Penicillin, Proc. Soc. Exp. Biol. & Med. 51:386, 1942.

6. McKee, C. M., and Houck, C. L.: Induced Resistance to Penicillin of Cultures of Staphylococci, Pneumococci and Streptococci, Proc. Soc. Exp. Biol. & Med. 53:33 (May) 1943.

7. Schmidt, L. H., and Sealer, C. L.: Development of Resistance to Penicillin by Pneumococci, Proc. Soc. Exper. Biol. & Med. 52:353 (Apr. 1) 1943.

8. Kolondy, M. H., and Denhoff, E.: Reactions in Penicillin Therapy, J. A. M. A. 130:16 (Apr. 20) 1946.

9. Dubos, R. J.: The Mode of Action of Chemotherapeutic Agents, (Bull.) New York Acad. Med. 21:27 (Jan.) 1945.

10. Wolff, L. K., and Julius, H. W.: Action du Sulfanilamide in vitro et in vivo, Ann. Inst. Pasteur 62:616, 1939.

11. Hobby, G. L., Meyer, K., and Chaffee, E.: Activity of Penicillin in Vitro, Proc. Soc. Exp. Biol. 50:277, 1942.

12. McKee, C. M., and Rake, G.: Activity of Penicillin Against Strains of Pneumococci Resistant to Sulfonamide Drugs, Proc. Soc. Exp. Biol. 51:275, 1942.

13. Spink, W. W., Ferris, V., and Vivino, J. J.: Comparative in Vitro Resistance of Staphylococci to Penicillin and to Sulfathiazole, Proc. Soc. Exp. Biol. 55:207, 1944.

14. Menkin, V.: The Mechanism of Fixation by the Inflammatory Reaction, J. Exp. Med. 53:171, 1931.

15. Menkin, V.: Fixation of Bacteria and of Particulate Matter at the Site of Inflammation, J. Exp. Med. 53:647, 1931.

16. Menkin, V.: Studies on Inflammation, J. Exp. Med. vol. 51, 1930.

17. Spink, W. W., Rantz, L. A., Boisvert, P. J., and Coggeshall, H.: Sulfadiazine and Penicillin for Hemolytic Streptococcus Infections of the Upper Respiratory Tract, Arch. Int. Med. 77:260 (March) 1946.

18. Mutch, N., and Rewell, R. E.: Penicillin By Inhalation, Lancet 1:650-652, 1945.

XXVI

NASAL PHYSIOLOGY IN RELATION TO THE COMMON COLD

ARTHUR W. PROETZ, M.D.

ST. LOUIS, MO.

Man has finally split the atom, but the common cold still eludes him. As our knowledge of the processes which take place in the normal nose gradually takes shape, however, certain symptoms which accompany its onset assume a new meaning.

First comes the indication that there are at least four, and probably more, different ways in which common colds begin—an important consideration in the choice of treatment.

Second, it seems equally likely that some of the disagreeable symptoms which we regard as the early phases of the cold are actually not a part of the infection at all, but are separate phenomena which set the stage for the infection and are therefore of utmost importance in prevention.

As a background let us review briefly the conditions which exist in the normal nose, admittedly a rare object but at least a starting point.

The upper air passage consists of a rigid, fairly tubular airway with which numerous small chambers communicate through still smaller openings. The whole structure is lined with a thin but tenacious coat of mucus which is kept in constant motion from nostril to pharynx by the cilia. While these are absent in the small olfactory and preturbinal areas, the mucus over the areas is nevertheless kept sliding over them by the surrounding cilia. The whole mucous blanket is propelled backward until it reaches the upper portions of the nasopharynx where it is taken over by the muscles of deglutition and sent on its way into the esophagus.[1, 2]

Let us take up our position by the side of a single ciliated epithelial cell somewhere in the nose, thus better to see what goes on. For convenience we will designate it Cell X.

Presented before the American Laryngological, Rhinological and Otological Society, Chicago, Ill., May 28, 1946.

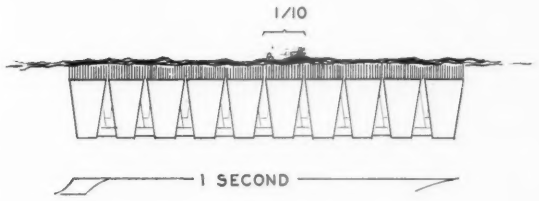


Fig. 1.—Diagram; profile of epithelial cells, illustrating average distance traversed by bacteria and particulate matter in 1 second.

Together with its millions of neighbors Cell X is topped by a stiff brush of cilia moving in propulsive strokes, a dozen to the second in the direction of the pharynx. Over it the blanket of mucus is travelling at the rate of ten times the diameter of the cell every second. The bacteria and other foreign matter are riding on the surface of the mucus, not touching the cell or its cilia. Obviously, so long as this mechanism remains intact throughout the nose no infection can take place, for no bacterium can remain in contact with our Cell X long enough to do it harm.

For infection to take place something must occur which permits the invading organism to remain in contact with an individual cell long enough to permit penetration. Four such agencies will be discussed, two acting directly on the mucosa and two remotely:

1. Structural mechanical obstruction.
2. Drying.
3. Functional mechanical obstruction.
4. Biochemical stasis of the mucous blanket.

First. A deviated septum, a hyperplastic turbinate, an adhesion, a polyp, a foreign body or a scar—any of these can by their mechanical characteristics block the passage of the mucous blanket and arrest bacteria long enough to permit penetration. If the germs are virulent severe infection takes place but usually the infection is local. The obstruction being fixed, these minor infections recur frequently, metaplasia occurs (the presenting surfaces of polyps for example are usually not ciliated) and the area subsides into a chronic nuisance rather than a source of colds. Agency 1. is thus not frequently a common cold producer.

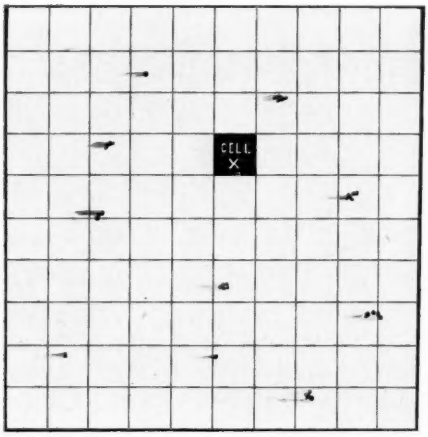


Fig. 2.—Diagram; surface view of epithelium. Bacteria crossing from left to right traverse the field in 1 second. Cell X will be crossed by the bacteria at its left in one-tenth second.

Second. Concentrated jets of dry air can render the mucous blanket at some point first sticky, then dry, not only stopping its progress but drying out and destroying the ciliated surface cells. At these points of stasis there is time for invasion if pathogenic bacteria are present. If the flora is unpropitious, colds occur. These dry spots usually occur in the same locations in a given individual. Such patients know when they are taking cold because the familiar hot-spot is on fire.

These dry spots are caused by restricted or distorted airways projecting air in concentrated streams. The restrictions and distortions may be due to fixed nasal abnormalities or they may come about through sudden vascular swellings and obstructions resulting from bodily changes described in the following category.

Third. This is the most common cause of endemic winter colds, and comes about as follows: *a*) Something occurs in the body to upset the sympathetic system—sudden chilling, fatigue, constipation, hunger, excitement or the ingestion of alcohol. The patient may be uncomfortable but still has no cold. *b*) The nasal erectile tissues susceptible to vasomotor stimuli react; they may shrink slightly at first but soon swell, causing obstruction and diversion of air currents. The concentrated dry air jets make burning dry spots usually

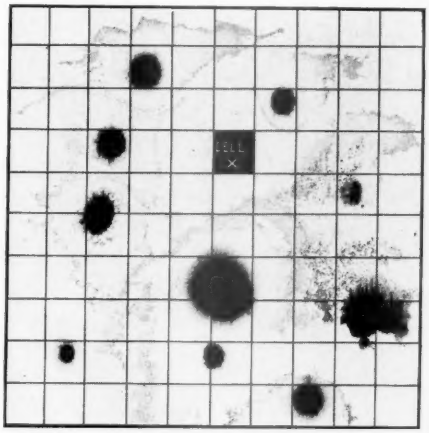


Fig. 3.—The diagram shown in Fig. 2 was moistened and a soluble pigment was substituted for the "bacteria". The relatively wide spread effect is obvious.

in the nasopharynx. There is still no infection—no cold. *c*) The stasis in the nasopharynx permits accumulation and growth of bacteria at a point where lymph glands are superficial and susceptible. Only now for the first time bacteria penetrate and unless promptly phagocytosed pass downward in the lymphatics. Systemic toxic symptoms appear. The vasomotor system is now doubly upset. The nose blocks up tight. Nasal retention of any organism which may be there takes place. The cold is in full swing.

This is a familiar clinical picture. In a given patient the attacks usually begin in the same location with a sharp burning in the pharynx or the nasopharynx, some 24 hours before the onset of the nasal disturbance. This danger signal gives us several hours in which to adopt measures for aborting the attack before it establishes itself in the lymphatics and involves the nose. Treatment now depends upon the re-establishment of normal conditions as quickly as possible and the scrupulous avoidance of any measures which will upset the still functioning nasal mechanism. One can easily wreck the whole procedure at this point by irrigating away the highly essential mucus or by applying strong constrictors which increase the nasal caliber and therefore the stickiness and unmanageability of the mucus in the nasopharynx.

Fourth. There may be a direct attack by a biochemical agent, presumably soluble. This type of onset is one in which the cilia are primarily incapacitated — directly overwhelmed — by the invading agent. As pointed out in the beginning, no organisms lightly scattered over the surface can remain in contact with any one cell long enough to accomplish this. Therefore, the invader in this type of cold must either be applied to the surface in large quantities at one time—an unlikely occurrence— or it must be soluble in the nasal mucus and thus be distributed over areas large enough to permit the prolonged exposure of a given cell. Virus infections may well be of this nature.

Authors discussing this mode of attack are apt to deal with it briefly, mentioning only that the cilia are incapacitated or "paralyzed". We have seen paralysis going on under the effects of cocaine or epinephrine or intense cold (10° C.) but under no other agency. Since the reacting agent in the case of the common cold is unknown, it is difficult to set up an experiment in the clinic or the laboratory to observe its action.

At a recent meeting of the American Association for the Advancement of Science, Professor James A. Harrison of Temple University presented a motion picture dealing with certain antigen-antibody reactions in paramecia.[3] As the antigen began to take effect the paramecia which had been swimming about freely slowed down (their courses becoming erratic) and finally came to rest. It could be seen in the photographs that this was due first to an amorphous mass, presumably jelly-like, which appeared on top of the cilia and prevented their propulsion of the animal by forming an envelope about it. At least in the initial stages the activity of the cilia beneath the envelope was in no way diminished.

Professor Harrison's experiment suggests the possible mechanism of the so-called "paralysis" of the cilia in the fourth type of cold. It may explain the characteristic failure of certain severe epidemic colds to respond to any treatment, no matter how vigorous or how promptly begun. The logical approach then would be a method of preventing, dissolving or washing away the envelope or the presumably soluble invading substance giving rise to it.

We have attempted to reproduce Professor Harrison's phenomenon on the ciliated mucosa of living animals and upon extirpated specimens of epithelium but so far have been unable to set up an experiment which meets the conditions and at the same time permits us to observe to our satisfaction.

The purpose of this presentation has been to try to put into some concrete classification compatible with the physiology of the nose what is known regarding the onset of colds in their various guises. Whether all the details of the classification are correct is of less importance than 1) that colds arise in different ways, 2) that they are apt to repeat a pattern in a given individual, affording us the opportunity of anticipating them, 3) that in many instances the prodromal signs indicate means of preventing the attack and, 4) that an investigation of the means by which cilia and their mucous blanket are incapacitated is likely to open the way to more effective prevention of epidemic colds.

BEAUMONT BUILDING.

REFERENCES

1. Sluder, G.: Die Wechselbeziehungen zwischen Aktion des Pharynx und des weichen Gaumen, etc., Arch. f. Laryng. u. Rhin., 1915.

2. Sluder, G.: Correlated Action of Pharynx and Soft Palate, Trans. Amer. Laryng. Assn., 1914, p. 142.

3. Harrison, James A.: A Serologic Study of Conjugation in Paramecium Bursaria, J. Exper. Zool. 101:425-444 (April) 1946; and personal communications.

XXVII

MALIGNANT TUMORS OF THE MAXILLARY SINUS:

AN ANALYSIS OF 47 FATALITIES

JOHN J. O'KEEFE, M.D.

AND

LOUIS H. CLERF, M.D.

PHILADELPHIA, PA.

Review of recent writings[4, 6, 7] substantiates the fact that the diagnosis of malignancy of the paranasal sinuses all too often is made during the late stages of the disease. This is due in great measure to the fact that patients suffer the presence of tumor masses of the face, paresthesias, dental pain, and infraorbital pain for prolonged periods, before seeking medical care. Another reason, and an alarming one, is the degree of procrastination played, both on the part of the physician and on the part of the patient, before definitive therapy is instituted.

This summary is a study of 47 such cases observed in the Radium Clinic of the Philadelphia General Hospital. When initially presented, all showed evidences of extension of the primary antral disease, and in spite of intensive and variously combined forms of therapy, all resulted in death.

As with malignant disease in other parts of the body, a single primary cause is not known. Many contributing or predisposing factors have been ascribed, but except for the factor of chronic irritation, none hold anything in common.

The antra, especially protected as they are from the ordinary traumatic agents of the respiratory tract, are influenced less by such inhalation irritants as exhaust gases and tobacco smoke than are the other parts of the upper respiratory tract. Silica, a frequently found co-incident in bronchogenic carcinoma, plays no role in sinus malignancy.

In all likelihood, the paramount chronic irritant of sinus malignancy is that associated with infection. But again, as is evident clinically, although this is a common contributing factor, its absence

From the Department of Radiology, Philadelphia General Hospital.

is noted too frequently to term it a basic etiologic factor. It should follow, then, that malignant disease of the sinuses may be the result of a pecular type of metaplasia associated with the repair of epithelium destroyed by disease. Evidently this statement is not all inclusive, for such accidents as cell rests, dermoids, and epithelial extensions must be given equal consideration.

Males generally are more frequently observed with carcinoma of the sinuses, and in this study there were 33 males to 14 females, or a proportion of 70.2 per cent male to 29.8 per cent female.

TABLE 1.—AGE AND SEX DISTRIBUTION.

AGE GROUP	MALE	FEMALE
21-30 years	1 (2%)	0
31-40 years	5 (11%)	1 (2%)
41-50 years	4 (9%)	5 (11%)
51-60 years	8 (17%)	4 (9%)
61-70 years	10 (21%)	4 (9%)
71-80 years	3 (6%)	1 (2%)
81-90 years	1 (2%)	0

The age distribution is of wide variation, but the highest incidence occurs in the age groups between 50 to 70 years. Twelve or 25.5 per cent were in the 51 to 60 year group, and 14 or 29.7 per cent were in the 61 to 70 year group. The youngest patient was 26 years old, and the oldest 85 years. The relative slight increase in age incidence noted here is due, we believe, to the fact that these cases were seen late in the existence of the disease.

The duration of the patients' chief symptom, when first seen, varied from one month to 28 years, and all complained of multiple symptoms prior to seeking medical care. In the order of frequency, palpable swelling of the face, or bulging of the eye, was most frequently complained of, closely followed by pain in the face or teeth. Table 2 lists these complaints in numerical sequence:

TABLE 2.—SYMPTOMS ON ADMISSION TO THE HOSPITAL.

Swelling of face	44.6%
Swelling of eye	17.0%
Pain in face	31.9%
Pain in eye	12.7%
Epistaxis	27.6%
Nasal obstruction	12.7%
Amblyopia	8.5%
Cervical adenopathy	8.5%
Headache	8.5%
Paralysis of face	2.1%
Paralysis of body	2.1%
Paralysis of larynx	2.1%

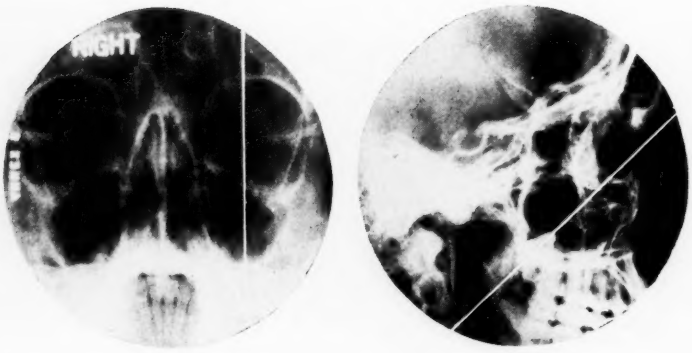


Fig. 1.—Regional classification of the antrum into Malignancy Areas as suggested by Ohngren. *A.*—Vertical line dividing the antrum into medial and lateral halves. *B.*—line extending from the inner canthus of the eye to the angle of the jaw dividing the antrum obliquely.

An analysis of the time intervals, between the onset of the original symptom and hospitalization illustrates the amount of time lost before treatment was sought. It also evidences the need for such a regional classification of the antrum into malignancy areas as suggested by Ohngren.[1]

TABLE 3.—TIME BETWEEN INITIAL SYMPTOMS AND HOSPITALIZATION.

13 cases	(27.6%)	more than one year
12 cases	(25.5%)	six months to one year
22 cases	(46.8%)	less than six months

He has projected a line onto the antrum, extending from the inner canthus of the eye to the angle of the jaw, thereby bisecting it obliquely into superior and inferior divisions. Another line, dropped vertically through the center of the antrum, further subdivides these areas into medial and lateral halves. The rationale of this is sound, because by its use one is kept mindful of the wide differences in rates of growth and comparative responsiveness to treatment of the two areas, and too, because therapy based on it may be more accurately decided upon.

Those cases listed as being present for more than one year have had their origin in the dormant or slow growing area—the lateral

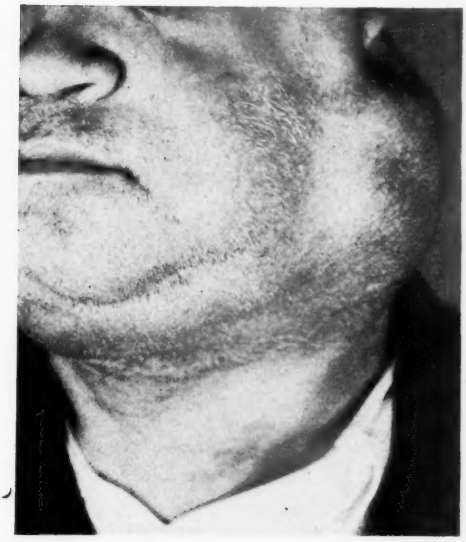


Fig. 2.—Carcinoma of the antrum with massive involvement of the regional cervical lymph nodes. Inoperable.

part of the superior division. Those present for less than a year were faster growing and originated in the medial part of the superior division and in the inferior division.

According to Snitman,[2]

"Lesions above the Ohngren plane, situated medially, are frequently associated with nasal polyps, and encroach on the meninges more readily and tend to early lymphatic invasion. In the lateral part of the suprastructure, carcinoma has at its onset the most dormant development. It then rapidly invades the malar bone producing the characteristic tumefaction of the external angle of the floor of the orbit.

"Malignancies in the infrastructure of the antrum produce earlier symptoms referred to the teeth, but these symptoms are still overlooked until late, because attention is directed to the dental structure for a considerable period of time."

Such anatomic or regional classification is possible even in late cases, conceding the fact that the area of bone destruction is the site of the original disease. Using such classification, results of various forms of therapy may then be more accurately tabulated and evaluated.

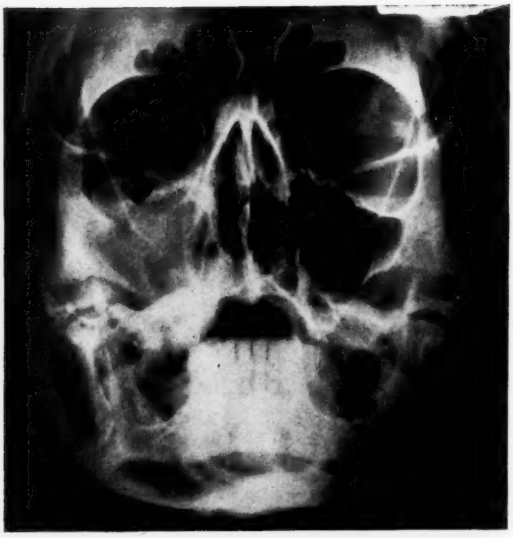


Fig. 3.—Roentgenogram illustrating destruction of the floor of the orbit.

One other factor is essential to the proper and complete evaluation of malignant disease of the antrum: the presence or absence of regional lymph node extension.

These cervical chains act as protective barriers to the distant spread of cancer cells, as ably testified by Braund and Martin,[3] who report metastases below the clavicle in only 23 per cent of a series of 284 autopsies, wherein death resulted from cancer of the upper respiratory and food tracts.

In only four cases in this series was cervical adenopathy the initial symptom; however, it was present with other symptoms in the majority of patients when first seen. Conversely, paralyses, the effects of remote metastases, were initially evident in only three.

The diagnosis of malignant disease of the maxillary sinus, especially in late cases, is many times so obvious that it is tantamount to a clinical entity. Early lesions, on the other hand, largely because of their asymptomatic growth, entail great difficulty in diagnosis. For

these we must rely on the adjuncts of the roentgenogram and histopathologic studies.

In addition to the clinical picture, supportive proof of malignancy was made in every instance—either by x-ray examination or by biopsy, and in several cases, by both. A comparative study of these two methods presents an interesting picture, but unless properly evaluated and explained, may be misleading.

TABLE 4.

X-RAY EVIDENCE OF CARCINOMA		HISTOLOGIC EVIDENCE	
By bone destruction	29 or 61.7%	Positive	21 or 44.6%
Increased densities	6 or 13.7%	Indefinite	2 or 4.25%
No report	12 or 25.5%	Chronic inflammation	4 or 8.5%
		No report	20 or 42.5%

An added source of information was gained from those who had regional extensions. Needle aspiration, and occasionally the resection of superficial nodes, ofttimes supplied the information necessary to diagnosis when other means were indefinite or lacking. Such extensions were present in all 47 of these cases, either in the form of intranasal tumors, cervical nodes, or intra-oral lesions.

Regarding the pathology of these malignancies, we quote from Eggston's[4] monograph,

"Malignant epithelial tumors of the upper respiratory region may be conveniently described under their morphologic appearance histologically:

Epidermoid
Papillary carcinoma
Adenocarcinoma
Mixed salivary gland (malignant)

"The epidermoid carcinoma or epitheliomatous type is by far the most common type of malignancy of the nose and oropharynx. . . . In the diagnosis we have found it convenient to use Broders'[5] classification as to type. If three-fourths of the cells present are differentiated, we have called it roughly type I. If one-half of the cells revert to the parent type, the tumor has been classed as type II. If approximately one-fourth of the cells are differentiated, it has been classed as type III. If there is marked anaplasia and practically no attempt at reversion to the parent type, the tumor has been classed as type IV. Such a classification may be of some importance in determining the embryonic character of the growth and at times appears to be of practical use in the evaluation of the ray sensitivity of the tumor.

"Squamous carcinomas of the nose and nasal sinuses usually are preceded by a metaplasia of columnar epithelium to the pavement type. This metaplasia follows chronic irritation or infection of the antrum or other sinuses. There may be, on the other hand, extension of squamous cells from a fistula or tooth socket, the cells may be a part of a dermoid cyst, or there may be some misplacement of a squamous-cell rest in the complicated embryological development of the nasal structure."

TABLE 5.—TYPES OF TUMORS IN 21 POSITIVE BIOPSIES

	No.	%
Squamous Cell		
Type II	5	23.8
Type III	6	28.5
Type IV	6	28.5
Adenocarcinoma	2	9 5
Mixed Tumor	1	4.7
Sarcoma	1	4.7

It might well be interpolated, relative to this discourse, to say that it not only provides a basis for classification of carcinoma on an embryologic basis, but that by virtue of its very elemental principles, throws some light on the possible etiologic factors.

New and Erich[6] state that "the successful treatment of malignant neoplasms of the nose and sinuses, is based on early diagnosis and proper management." They further reiterate that an early diagnosis is "extremely important." Other factors influencing management are the proper graduation of the tumor as outlined by Broders, the regional classification of the site of origin, the presence or absence of regional metastic nodes in the cervical areas, and the age and general physical condition of the patient.

Recent developments[7, 8] have shown that, in general, low-grade carcinomas are relatively radio-resistant, and good results are often obtained with the use of surgical diathermy. On the other hand, high-grade tumors being radio-sensitive respond better to irradiation.

Such rationalization applies in theory; not often in fact. Practically, one cannot determine the degree of extension beyond the confines of the antral wall, and to limit the management to electrosurgery or to radiation, in any given case, would be to deprive the patient of all advantages in the attempt to secure a cure of his disease.

The combination of electrosurgery and irradiation—either radium or the roentgen ray, or both—is the ideal practice and produces the best results in unselected cases. Del Regato[9] reports several cures by the use of protracted external irradiation alone.

The problem of metastatic cervical lymph nodes should not influence the management of the antral lesion. If metastases are present, they constitute a problem unto themselves. If operable and cure of the antral disease is likely, they should be resected, followed by fractional external irradiation. If inoperable, irradiation, either by external application or radon seed implantation, or both, should be given.

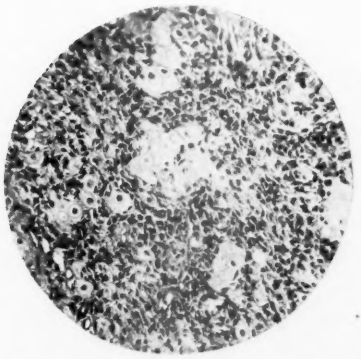
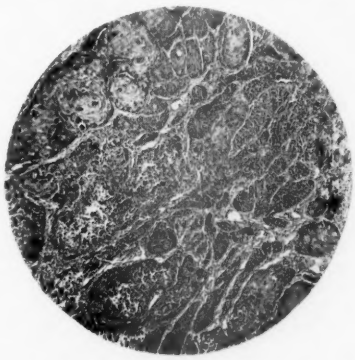
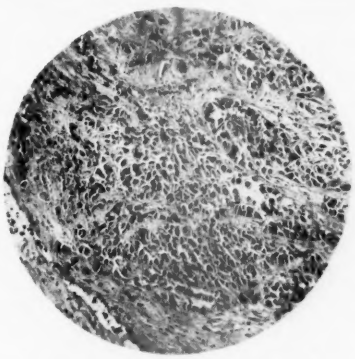


Fig. 4.—A.—Photomicrograph disclosing very irregular cartilage-like cells surrounded by spindle cells resembling fibrosarcoma. x 100.



B.—Photomicrograph showing squamous cell carcinoma, Type II. x 38.



C.—Photomicrograph showing anaplastic squamous cell carcinoma, Type IV. x 100.

The old and debilitated patient, unable to stand the shock of extensive surgery or intensive radiation, and those having hopelessly extensive lesions, are best treated palliatively. Cure, in these instances, is not looked for.

Death is usually the result of the "cachexia of carcinoma." Progressive extension of the primary and metastatic lesions, ofttimes associated with sepsis and intermittent hemorrhage, and complicated by pneumonia from aspiration, produces a rapid decline in the patient's physical state. Occasionally in the older groups, a merciful

TABLE 6.—CAUSES OF DEATH.

	No.	%
Extension of primary and metastatic lesions (sepsis, pneumonia, etc.)	37	78.7
Massive hemorrhage	6	12.7
Coronary thrombosis	1	2.1
Refused treatment	1	2.1
Not related to cancer (cures?)	2	4.2

coronary occlusion eventuates. Tabulation of the exact cause of death is an indefinite process, generally because such terms as "debilitation" and "inanition" are so nonspecific. Table 6 is a gross attempt at such a classification, keeping in mind that the fundamental cause is carcinoma.

TABLE 7.—LENGTH OF LIFE AFTER ADMISSION.

18 cases (38.3%) less than 3 months
10 cases (21.2%) 3 months to 6 months
12 cases (25.5%) 6 months to 1 year
5 cases (10.6%) 1 year to 2 years
2 cases (4.2%) more than 5 years*

*These are the same two whose deaths are reported as "not related to carcinoma," in Table 6.

SUMMARY AND CONCLUSION

1. Forty-seven cases of antral malignant disease resulting in death are presented. In that all cases were seen late in the existence of the disease, the factor of early diagnosis is stressed.

2. Classification of malignant disease of the maxillary sinus should be determined as completely as possible, and should include the duration of symptoms, the clinical manifestations, the embryonic type of cell, the regional site of origin, and all predisposing factors.

Evaluation of this classification should be the basis for the variations in management.

3. Newer methods of treatment are securing better results in selected cases. The combined use of electrosurgery with radiation offers the best chance of cure; protracted fractional external radiation is a valuable supplement.

255 South Seventeenth Street

1530 Locust Street.

REFERENCES

1. Ohngren, L. G.: Malignant Tumors of the Maxillo-ethmoidal Region: Clinical Study with Special Reference to Treatment with Electrosurgery and Irradiation, Acta Oto-laryng. (Suppl. 18) 171-174, 1933.

2. Snitman, M. F.: Individualization in the Management of Carcinoma of the Maxillary Sinus, Annals of Otology, Rhinology and Laryngology 54:125-135 (March) 1945.

3. Braund, R. R., and Martin, H. E.: Distant Metastases in Cancer of the Upper Respiratory and Alimentary Tracts, Surg., Gynec. and Obst. 73:63-71 (July) 1941.

4. Eggston, A. A.: Histopathology of the Nose and Throat, in "Surgery of the Nose and Throat", edited by John D. Kernan, New York, Thomas Nelson and Sons, 1942.

5. Broders, A. C.: Carcinoma: Grading and Practical Application, Arch. Path. 2:376, 1926.

6. New, G. B., and Erich, J. B.: In Jackson and Jackson, Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1945.

7. New, G. B.: Malignant Tumors of the Antrum of Highmore; End-Results of Treatment, Arch. Otolaryng. 4:201-214, 1926.

8. New, G. B.: Malignant Diseases of Paranasal Sinuses, Am. J. Surg. 42:170, 1938.

9. Del Regato, J. A.: Roentgentherapy in Epitheliomas of the Antrum, Surg., Gynec., and Obst. 65:657-665 (Nov.) 1937.

XXVIII

ADAMANTINOMA OF THE MAXILLARY SINUS WITH A REPORT OF TWO CASES

CLYDE A. HEATLY, M.D.

ROCHESTER, N. Y.

Adamantinoma occurs far more frequently in the mandible than in the maxilla. Involvement of the maxillary sinus by this tumor is uncommon in the experience of most rhinologists. When first encountered, therefore, the true nature of the neoplasm may not be recognized and inadequate surgical measures may be employed. The clinical picture furthermore may be mistaken for a malignant growth of the antrum and the microscopic examination in cases of the undifferentiated types of this tumor may be misleading. The purpose of this presentation is to review briefly the pathological and clinical characteristics of the adamantinoma and to discuss the problems concerned in its removal.

Adamantinoma may be defined according to Robinson[1] as "an epithelial tumor arising from the odontogenic apparatus or from cells with a potentiality for forming tissues of the enamel organ." The term adamantinoma, introduced by Borst in 1902, is obviously misleading inasmuch as this tumor never forms enamel (adamantine) tissue. The term adamantoblastoma has been suggested by Thoma[2] as being more accurate in this respect and Robinson and Churchill refer to the tumor as ameloblastoma, deriving the term from ameloblast, the enamel forming cell. The name adamantinoma will be retained in this paper because of its common usuage and wide familiarity. This tumor is classified with the epithelial cystic odontomas which also include dentigerous and dentoperiosteal cysts.

Regarding their pathogenesis the most widely accepted view at present is that adamantinomas arise from paradental epithelial rests. Malassez first observed cordlike and glandlike groups of epithelial cells around the roots of the teeth and referred to them as "paradental epithelial debris." He further demonstrated that these cells

---

From the Department of Surgery, Division of Otorhinolaryngology, The University of Rochester School of Medicine and Dentistry, Rochester, New York.

Read before the meeting of the American Laryngological Association, Chicago, Ill., May 30, 1946.

may remain as scattered epithelial rests even late in life. Under certain irritative conditions these cells become proliferative and give rise to tumor formation. The site of predilection of the adamantinoma in the region of the lower third molar, the most difficult of the teeth to erupt, is pointed out by New[3] as evidence of the important influence of irritation. It is a clinical fact that many reported cases record preceding traumatic or irritative factors in the form of dental extraction, injury to the jaw, impaction or infection.

The adamantinoma may be described as a central tumor, slow and intermittent in its growth, anatomically benign but clinically persistent. Macroscopically two types are recognized, the solid and the cystic. Most authorities agree with Kronfeld that the adamantinoma is originally a solid growth and that the cystic variety represents a later degenerative process occurring within the epithelial strands of the tumor. Although monocystic forms occasionally occur, most tumors are of the polycystic variety. In a series of 219 cases reviewed by Robinson 57.5 per cent were cystic, 24.2 per cent were both cystic and solid and 19.1 per cent were solid. It is important in this connection to point out that such tumors involving the maxilla are more often of the solid variety than those found in the mandible. The solid tumors are usually of a white, finely granular consistency, containing connective tissue trabeculae and small cavities and are definitely encapsulated. In the cystic types, compartments of varying size may be noted, some filled with solid tumor and others with a thin yellowish fluid. Fibrous or bony septa can be seen separating the various cysts. Teeth are frequently found in these tumors and may cause a mistaken diagnosis of dentigerous cyst. Microscopically, the solid areas consist of a normal connective tissue stroma and columns of epithelial cells which may be elongated, rounded or arranged in acini-like forms. These cells resemble in varying degree the epithelial structures seen in the embryonic stage of the normal tooth. The peripheral layer is composed of a continuous palisade of columnar cells (ameloblasts) arranged in a single row upon an outer basement membrane. The middle layer is made up of vesicular cells and the center of the cell nest is composed of stellate cells representing the central stellate cells of the enamel organ. The stellate cells undergo disintegration and gradually disappear, leaving cysts containing fluid and lined with the outer columnar cells. In the larger cysts the walls consist of fibrous tissue only. Authorities agree that no information can be gained from the microscopic picture concerning the malignant possibilities of the tumor although Ewing[4] considers the solid tumors to be more malignant than the cystic types.

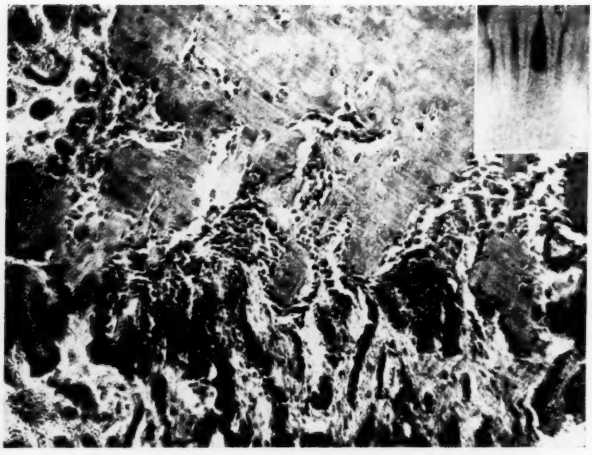


Fig. 1.—Section of alveolar bone surrounding an adamantinoma showing finger-like extensions of the tumor into the bone. (From Thoma[2] with permission of the author.)

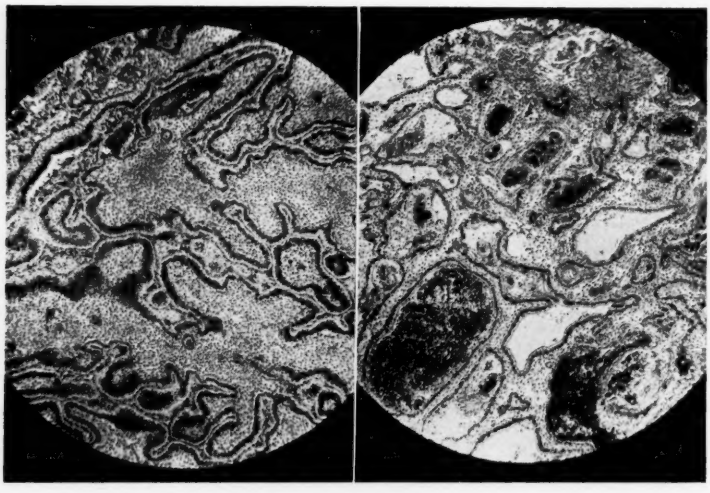


Fig. 2, Case 1.—Adamantinoma of the maxillary sinus (solid type).

Fig. 3, Case 2.—Adamantinoma of the maxillary sinus (polycystic type).

Pathologists commonly classify the adamantinoma as a benign neoplasm. As Havens[5] and other authors have emphasized, its relentless growth, its persistent recurrence if incompletely removed and finally the evidence of several proved instances of metastasis indicate that for clinical purposes at least it should be regarded as a malignant growth. Microscopic study of the bone surrounding this tumor frequently shows proliferation of the tumor cells in the spongiosa or the haversian system of the cortex. Both Thoma and Havens have stressed this important fact and presented illustrations of the microscopic finger-like extensions of the tumor into the surrounding bone (Fig. 1). Robinson, in his review of 379 cases from the literature, found 17 cases of malignant adamantinomas, an incidence of 4.5 per cent. Both carcinomatous as well as sarcomatous changes have been reported. One rare case of melanotic adamantinoma was described in 1926 by Mummery and Pitts. For many years it was believed that this tumor does not metastasize. There are now on record however several accepted cases of metastasis to regional glands or internal organs. Schweitzer and Barnfield[6] in 1943 after a careful review of the literature were able to collect ten reports of metastasis from adamantinomas, although in their opinion four of these were not acceptable. They also presented a case occurring in the mandible of a young negress which recurred locally despite 24 operations and finally metastasized by the blood stream to the lungs. Vorzimer and Perla in 1932 described an adamantinoma in the maxilla of a 17-year-old boy which metastasized by the airways to the right lung and pleura. Phelps[7] in 1939 reported a case of malignant adamantinoma with metastases to the liver, lungs, ribs and clinically to the left retina. Chont[8] in 1943 reported eight cases of adamantinoma in one of which metastases from a tumor in the mandible occurred in the cervical glands and lungs. Metastases to the submaxillary glands and cervical lymph nodes have been reported by New, Havens, Horsley, Simmons, Weissenfels and others. It must be concluded therefore that while the adamantinoma characteristically grows slowly by expansion rather than by infiltration and does not metastasize, malignantly infiltrative varieties as well as true metastasis may occur.

Adamantinoma may occur at any age. It has been reported in early childhood and, by Lewis, in a man of 75. In Robinson's survey of 379 cases, the average age at the time of observation was 37.6 years. The average age incidence in 114 cases reviewed by McFarland and Patterson[9] was slightly less than 40 years. The average duration of symptoms in Robinson's survey was eight and a half years and in McFarland and Patterson's, seven years. The average

age at the onset of symptoms therefore may be considered to be the early thirties. Murphy[10] observed that the solid variety of this tumor is more common in younger patients and the cystic type more frequent in later life. This observation is in accord with the characteristic cystic degeneration already described in the evolution of these tumors.

The majority of adamantinomas occur in the mandible. Eighty-three and seven-tenths per cent of 379 cases reviewed by Robinson involved the lower jaw. In a series of 58 cases reported by Havens from the Mayo Clinic only five were in the maxilla. In 114 cases reviewed by McFarland and Patterson, 17 involved the upper jaw. They considered the proportions to be roughly five to one. Interesting recent reports of this tumor involving the maxillary sinus have been made by Phelps,[7] Golden[11] and Mosher.[12] Ewing and Murphy both agree that solid adamantinomas are more frequent in the upper jaw and further that this location shows a greater incidence of the malignant forms.

The adamantinoma is a slow growing, centrally expansive tumor. In the upper jaw the relatively large dimensions of the maxillary sinus permit great expansion of the growth before its presence is clinically apparent. Early diagnosis is uncommon and ill-defined pressure pains are usually mistaken for dental causes. Roentgenograms in this early stage are inconclusive. Slow expansion of the facial and nasal walls of the maxilla with resulting deformity of the face and obstruction of the nasal passage is commonly the chief presenting symptom. At this stage the thin, parchment-like bony capsule, crepitating on pressure, is often noted. Pain is usually slight except in the more advanced tumors. Occasionally a cystic tumor may rupture into the mouth and the patient seek advice because of a persistent oral drainage. Extension into the orbit with exophthalmos and into the nasopharynx with dysphagia has also been reported.[4] Broadening of the alveolar ridge with displacement of the adjacent portion of the hard palate may occur.

The diagnosis of adamantinoma is suggested by the slow, frequently painless development of the tumor, by the absence of evidence of soft tissue invasion, by the crackling sensation which can commonly be elicited on palpation over prominent portions of the tumor, by the usual absence of regional glandular involvement (exceptions to this have been pointed out), and finally by the roentgenogram in which the multilocular cystic arrangement of the bony shell and trabeculae is characteristic. The tumor is often clinically mistaken for a malignant tumor of the antrum and the roentgenographic

signs of destruction of portions of the bony walls without evidence of trabeculation may strongly suggest this diagnosis. Such a malignant tumor, however, differs in the rapidity of its growth, the more common association of glandular metastasis, the frequent evidence of soft tissue invasion as well as the absence of crepitation. Dull, persistent facial pain is more common in malignant tumors. Dentigerous cysts may also prove confusing especially in view of the fact that teeth are frequently found in adamantinomas. Oesterreich in 1936 emphasized this point and reported three cases which were roentgenographically diagnosed as cysts but which on histologic examination proved to be adamantinomas. Ewing also recorded two instances of monocystic adamantinomas clinically mistaken for periodontal cysts. Adamantinoma in his opinion must "always be kept in mind even if the roentgen film shows a monocystic defect or if the cyst contains a tooth."

Regarding the prognosis, the adamantinoma must be regarded as a slow-growing expansive tumor with a strong tendency to recurrence. In Robinson's review of 379 cases, recurrences were reported in 119 cases in which from one to 22 recurrences were noted. Incomplete removal tends to disrupt the expansive growth of the tumor and induces a more malignant and infiltrating variety, which in the maxilla may eventually involve inaccessible and ultimately fatal locations. Recurrence is frequently caused by the spread of the tumor into the spongiosa or haversian system of the surrounding bony cortex as well as by small tumor rests not easily accessible to surgical removal. It is generally agreed that the prognosis in the solid type of tumor is not as favorable as in the cystic variety. Unilocular cystic tumors as well as multilocular tumors with few compartments are more easily completely eradicated than honeycomb tumors with numerous small scattered loculi. Ewing states that "tumors of the upper jaw are much the more serious and that the solid adamantinomas of all types recur locally and involve antrum, orbit and nasopharynx. In spite of their relatively benign structures, the prognosis in this group is unfavorable." All authorities agree that any adamantinoma in which the rate of growth is suddenly increased should be given a guarded prognosis because of the strong likelihood of malignant changes.

In the treatment of adamantinomas, complete and radical removal of the tumor is necessary because of the clinical certainty that recurrences will occur if small areas remain. Although the adamantinoma is properly considered as a benign tumor by the pathologist, it is better for the rhinologist to treat it as a malignant growth from

the outset and to employ radical measures for its complete removal. Perhaps the greatest difficulty in this respect is caused by the tendency of the tumor to proliferate into the spongiosa or the haversian system of the surrounding bony cortex. The literature is rich with examples of the failure of conservative measures. In the series of 166 cases collected from reported sources by McFarland and Patterson in 1931, there were 65 cases in which a variety of conservative methods had been employed and in this group recurrence was reported in 50.7 per cent. Simple curettement will seldom succeed in eradicating this tumor. Havens reporting in 1939 on the experiences at the Mayo Clinic urged radical resection with the meticulous use of surgical diathermy. Waldron[13] similarly stressed the value of surgical diathermy as the method of choice in accomplishing complete removal. The bony wall surrounding the tumor should be removed where possible or coagulated with the diathermy current. Tumor nests in the ethmoid cells should be sought out and carefully destroyed. Accessibility of the antrum for subsequent examinations should be made possible by the creation of a large window in the inferior meatus as well as by maintaining the opening in the canine fossa for a considerable period. This will also supply adequate drainage for the slough following electrocoagulation. Many writers, including Kotany, Quick and Chont advise the routine use of intensive postoperative irradiation. It is well recognized however that the adamantinoma is extremely resistant to radiation and it is doubtful in the opinion of the writer if the dangers of radio-osteonecrosis justify the slight possibility of help from this source. It must be admitted however that in extremely extensive or recurrent, invasive types of adamantinomas postoperative irradiation should not be withheld. Finally, periodic examination for possible recurrence must be continued for several years following operation in view of the fact, already emphasized, that the rate of recurrence may be exceedingly slow.

REPORT OF CASES

CASE 1.—H. M., a white Greek woman of 65, was admitted to the Strong Memorial Hospital on September 13, 1943. Two years before she had noticed a fullness of the right side of the face and difficulty in breathing through the right side of the nose. She was advised by her physician that a sinus infection was present and had had frequent nasal treatments without relief. During the previous year the right side of the face had become increasingly prominent and the right nasal passage completely obstructed. There was no history of regional pain.

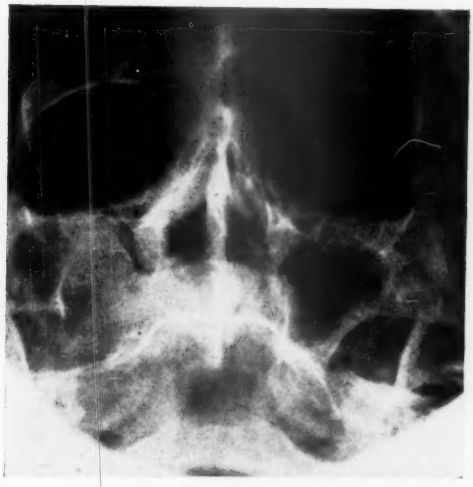


Fig. 4, Case 1.—Adamantinoma involving the right maxillary sinus. The true nature of the tumor can not be diagnosed from the roentgenogram.

On examination there was definite prominence of the right cheek with swelling of the lower eyelid. There was no evidence of soft tissue infiltration nor could any definite bony defect be palpated. No enlargement of the cervical glands was present. The entire lateral nasal wall was pushed over tightly against the nasal septum but no intranasal evidence of infection nor actual tumor invasion was present. The right antrum was completely opaque on transillumination. Roentgenograms showed the right antrum to be greatly enlarged and obscured by a shadow of rather uniform density (Fig. 4). No evidence of trabeculation could be seen. Destruction of the medial and lateral walls was noted together with thinning of the inferior orbital margin. This evidence indicated a tumor of the right antrum with considerable bony destruction.

A Caldwell-Luc operation was performed under sodium pentothal anesthesia. The anterior wall of the maxilla at the point of entrance was markedly thinned and easily entered. The antrum was found to be filled with a whitish, encapsulated tumor of rather firm consistency. A mass about 3 cm. in diameter was removed without any troublesome bleeding. The remainder of the tumor was then destroyed by coagulation. It was noted that this tissue pre-

sented a distinctly granular consistency. No large cystic areas were evident. The marked enlargement of the antrum with erosion of the medial and lateral walls noted in the roentgenogram was confirmed. All of the bony walls were greatly thinned. The ethmoid cells were cleaned out as thoroughly as possible through the antrum approach. A large window was resected into the inferior meatus and the canine fossa incision packed open.

Pathological Report. The gross specimen consists of smooth firm whitish tissue measuring about 3.5 cm. in diameter. It contains small cysts and masses of solid, and rather granular tissue. On microscopic study the tumor has a complex structure which consists of ramifying columns and masses composed of an outer single row of tall columnar epithelial cells arranged in palisade formation. The central areas are occupied by irregularly arranged stellate cells which stain lighter than the columnar cells. Numerous large and small cysts are seen; these are lined by columnar epithelial cells and contain either a pink coagulum or a delicate stroma of connective tissue and blood vessels. There are areas of fresh blood and numerous foci of phagocytes containing blood pigment (Fig. 2). Diagnosis—Adamantinoma.

The patient's postoperative course was uneventful and she was discharged from the Hospital on September 17, 1943. She was followed at two month intervals. A small recurrent tumor was observed on April 27, 1944, in the right middle meatus. This was thoroughly destroyed with the diathermy current. On September 5, 1945, another small mass in the inferior meatus was similarly coagulated. In both instances the recurrent tumor was about the size of a grape. No postoperative irradiation was given. This patient was last examined in April 1946 and showed no evidence of recurrence. The possibility of further recurrence, however, is considered likely in view of the solid nature of the tumor as well as its great size.

CASE 2.—C. M., a white American man of 65, was admitted to Strong Memorial Hospital on December 26, 1945, because of a draining fistula from the left antrum into the mouth. Twelve years before the patient had a wisdom tooth removed from the left upper jaw. The extraction was extremely difficult and the dentist reported that a fragment of bone came away with the tooth. The socket, however, finally healed after about four weeks of treatment. During the past year the patient had noticed a thin, slightly blood-tinged drainage in the mouth. Three months before admission a large opening was observed along the posterior portion of the alveolar ridge

and the drainage increased considerably in amount. At the time of entrance into the Hospital the drainage averaged about a tablespoonful a day. There had been no pain in the face at any time but some intermittent sense of fullness.

The important clinical findings were as follows: (1) Slight prominence of the left cheek. (2) A large draining sinus tract about 1 cm. in diameter in the approximate position of the left upper third molar. (3) Broadening of the alveolar ridge, slight but definite downward displacement of the adjacent hard palate and definite boggy crepitation on pressure over the palate as well as the anterior wall of the left maxilla. (4) No intranasal changes. (5) Completely opaque left antrum on transillumination. (6) No enlargement of the cervical glands. (7) Considerable expansion of the walls of the left antrum with evidence of destruction of the anterior wall and floor as shown by the roentgenograms. It was considered likely that we were dealing with an expansive and probably cystic tumor of the left antrum, possibly an adamantinoma.

A Caldwell-Luc operation was performed on December 28, 1945, under sodium pentothal anesthesia. Most of the anterior wall of the antrum as well as the greater portion of its floor was found to be reduced to eggshell thinness and at the point of entrance the bony wall had been completely destroyed. The antrum was filled with a whitish, granular tumor containing many small cysts. In the center of the mass was a large cystic cavity which communicated directly with the alveolar opening. The floor of the orbit as well as the lateral nasal wall was intact. The tumor tissue was meticulously removed and the ethmoid cells cleaned out thoroughly. No gross tumor tissue was found in the ethmoid cells. The bone of the anterior wall as well as the floor of the antrum and the lateral nasal wall was removed. The diathermy electrode was carefully used throughout the cavity. The alveolar opening was dissected out and closed. A large window was resected into the inferior meatus and the canine fossa incision packed open. The postoperative course was uneventful and the patient was discharged from the Hospital ten days later.

Pathological Report: One gram of friable, granular, whitish colored tissue is received. On microscopic study the tumor has a varied structure. In most areas the tumor consists of complex, branching columnar structures lined by a layer of tall columnar epithelial cells in palisade arrangement. The central portion of many of the columns contains pale, irregular stellate cells in varying stages of preservation. In others there are cystic spaces, some of which contain pale eosinophilic coagulum, while others are filled by fresh

and organizing blood. A few islands of squamous epithelium are seen. There are large areas of degeneration and hyalinization involving tumor cells and stroma. Many chronic inflammatory cells are present in these areas (Fig. 3). Diagnosis—Adamantinoma.

Comment: These two cases are examples of the solid and polycystic types of adamantinoma. The true nature of the tumor was not recognized in the first case at the time of operation, but was correctly diagnosed in the second instance. The experience gained in the first case as well as a better understanding of the recurrent nature of this tumor prompted a more radical plan of management in the patient. The possibilities, however, for recurrence in both cases are fully appreciated.

CONCLUSION

1. Adamantinoma involving the maxillary sinus should be treated as a potentially malignant tumor in view of its established tendency to recurrence if incompletely removed.

2. The likelihood of recurrence will be lessened by the removal of the bony antral walls where possible, as well as by the meticulous use of the coagulating current.

3. The early detection of recurrence will be aided not only by the creation of a large antral window but also by maintaining the opening in the canine fossa for a long period.

4. The value of postoperative irradiation is distinctly limited in view of the radio-resistant nature of this tumor.

5. The necessity for frequent reexamination of these patients for many years must be emphasized. The rate of recurrence is characteristically slow and malignant changes occasionally occur.

STRONG MEMORIAL HOSPITAL.

REFERENCES

1. Robinson, H. B. G.: Ameloblastoma: A Survey of 379 Cases From the Literature, Arch. Path. 23:831 (June) 1937.

2. Thoma, K. H.: Oral Pathology, St. Louis, C. V. Mosby Company, 1941.

3. New, G. B.: Cystic Odontomas, J. A. M. A. 64:34-39 (Jan.) 1915.

4. Ewing, J.: Neoplastic Diseases, Philadelphia and London, W. B. Saunders Company, 1940.

5. Havens, F. Z.: Benign Cysts and Adamantinomas of the Jaws, Arch. Otolaryng. 30:762-774 (Nov.) 1939.

6. Schweitzer, F. C., and Barnfield, D. D. S.: Ameloblastoma of the Mandible with Metastases to the Lungs: Report of A Case, J. Oral Surg. 1:287-295 (Oct.) 1943.

7. Phelps, K. A.: A Case of Malignant Adamantinoma of the Right Maxillary Sinus, Trans. Am. Laryng., Rhino., and Otol. Soc., 1939, pp. 393-395.

8. Chont, L. K.: Adamantinoma: Report of Eight Cases, Am. J. Roentgenol. 50:480-490 (Oct.) 1943.

9. McFarland, J., and Patterson, H. M.: Adamantinomata: A Review of One Hundred and Ninety-Six Cases Reported in the Medical and Dental Literature, Dent. Cosmos 73:656-670 (July) 1931.

10. Murphy, J. T.: Adamantine Epithelioma, Radiology 3:377-387 (Nov.) 1924.

11. Golden, M. G.: Ameloblastoma of the Left Maxillary Sinus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:378-388 (June) 1942.

12. Mosher, W. F.: Adamantinoma of the Maxillary Sinus, Arch. Otolaryng. 40:61-62 (July) 1944.

13. Waldron, C. W.: Tumors of the Upper Jaw, Surg. Gynec. and Obst. 72: 503-511, 1941.

XXIX

SCHWANNOMA OF THE PHARYNX

PIERRE VIOLÉ, M.D.

LOS ANGELES, CALIF.

Among other relatively rare developments, schwannoma of the pharynx takes a prominent place as an interesting example of benign tumor. Though this growth is not limited to the head and neck, these are the areas more frequently involved.

In a recent review of the subject by Ehrlich and Martin[1], covering a six-year survey at the Memorial Hospital in New York City, only 23 cases were encountered. In almost half of these the tumor was located in the head and neck, the neck being by far the most frequent seat of the growth. Since it is conceded to be a tumor of nerve origin, this frequency of occurrence in the cervical area is believed to be due to the presence of extensive peripheral nerve trunks in the neck.

In 1933 Figi[2] also stressed the infrequency of occurrence of primary neurofibroma of the pharynx, referring to the clinical and pathological study by New and Childrey[3] of pharyngeal tumors seen at the Mayo Clinic from 1917 to 1930. Of the scant 63 cases of benign tumor of the pharynx and tonsil observed in this period of years, not one primary neurofibroma in this region was encountered. As medical literature on this subject is strikingly lacking, the conclusion to be drawn is that this is due to extreme infrequency of occurrence. It is particularly rare preoperatively to find a typical Horner's syndrome associated with the tumor. In the case to be presented, as well as the one reported by Martin, the presence of a typical Horner's syndrome was evident. When accompanied by this syndrome, the additional symptoms characteristic of this complication may involve disturbance of the eyelids and other facial features, together with vasomotor and sudoral changes of the skin and face. This syndrome seems to be chiefly the result of involvement of the cervical sympathetic plexus, but may not entail all factors of the typical Horner's syndrome. Due perhaps to the greater frequency of neurosurgery, this syndrome is more commonly encountered than

---

Presented before the Western Section Meeting of the American Laryngological, Rhinological and Otological Society, Los Angeles, Calif., Jan. 26-27, 1946.

formerly. The increase in occurrence may be traceable to some extent to traumata from increased traffic and industrial accidents. The syndrome is also considered by some to be due to injury to the brain stem, to the spinal cord, or to the superior cervical ganglion or the nerve roots. Horner's syndrome may also result as a postoperative complication.

This growth would appear to be subject to occurrence at almost any age of the individual, and in either sex about equally. No common causative factor is recognized, though there has been some comment on the frequency of association with von Recklinghausen's disease.

These tumors invariably are found to be encapsulated, are usually smooth of surface, globular in form, though sometimes nodular, and unless degeneration of the tumor tissue has occurred, are found to be firm and fibrous. While the great majority of tumors of the schwannoma type have been observed to be smooth of surface, Hall and Owens[4] report a case of solitary neurofibroma of the pharynx showing extensive overgrowth of granulation tissue, which on inspection resembled carcinoma. However, microscopic examination revealed the typical neurofibroma which was removed with uneventful recovery.

When located in the neck, the tumor is usually anterior to the carotid artery and beneath the upper one-third of the sternocleidomastoid muscle. The tendency is to force the artery outward, with the tumor growing inward.

The size of the tumor varies—apparently in ratio to the length of time the growth has been in the process of developing. This process is invariably slow, sometimes extending over a period of many years before the patient is aware of the need of medical attention. While they sometimes attain large size before inspection, discomfort may be confined to slight throat irritation on swallowing. Since the growth originates in nerve tissue, it would logically lead one to expect pain as a characteristic symptom; however, pain is rarely noted, and then only when the tumor has attained sufficient size to cause increasing pressure against adjacent nerves, rather than being traceable to the nerve of origin. Difficulty in swallowing may persist, as in the case here presented, even after surgical removal of the growth, probably due to prolonged pressure on the cervical sympathetic plexus with irreparable nerve damage.

Formerly mobility horizontally and fixation vertically have been considered important signs in diagnosis; however, Ehrlich and

Martin discount to some extent the diagnostic value of these findings, as the same is true of other growths, such as carotid body tumors, with which this tumor might be confused.

Preoperatively, the tumor has much the same consistency and general appearance as the mixed tumors often encountered in this area. But on operation, it has been found that the schwannoma usually has a characteristic yellowish color, as contrasted to the grayish-white of most pharyngeal mixed tumors. Figi attaches some diagnostic importance to this color distinction. However, as stated by him, the problem of exact differential diagnosis must of course depend largely upon histological findings.

On sectional study, the surface of the tumor is glistening, the stroma of poor vascularity, though the capsular substance may be well supplied with blood vessels. Under the microscope this tumor reveals characteristic histologic features. The nuclei appear in palisade-like, parallel arrangement, usually elongated, and tend also to form in streams and eddies.

There has been considerable difference of opinion in the past as to whether the tumor is of ectodermal or mesenchymal origin. A tumor of the sheath of peripheral nerves was first described by Verocay[5, 6] in 1908 as "neurinoma." It has since become the concensus that this growth originates in the cells of the neurilemma or sheath of Schwann, and that it is therefore an ectodermal neoplasm. Thus the term "neurilemma", as contributed by Stout,[7] may be considered more precise. Coates,[8] in reporting a tumor of the mouth, states that he used the term "schwannoma" chiefly because the pathologist had used that term, although histologically this type of tumor is identical with the "neurofibroma" or "neurinoma", and that it is not limited to the oral area but may be found on any peripheral nerve in the body.

As reported by Ehrlich and Martin, experimental schwannomas recently have been produced by Masson,[9] who has traced both the experimental and spontaneous tumors back to the Schwannian theory of origin. Masson, and Murray and Stout[10, 11] have demonstrated with tissue cultures that the cells of these tumors are morphologically and physiologically similar to the cells of Schwann.

Turchik[12] reports a case of schwannoma of the pharynx which resulted in paralysis of the right vocal cord five years prior to discovery of the tumor. It conformed to the usual characteristic picture when seen for diagnosis, and was accompanied by Horner's syndrome postoperatively, which condition was still present after ten months.

In his opinion, Turchik considers this syndrome attributable to secondary inflammatory reaction of the sympathetic plexus, due to pressure of the tumor with resultant nerve damage. He expresses the belief that early diagnosis and operation should avoid the troublesome complications involved in this delayed case.

As is the case with carotid body tumor, only a tentative diagnosis can be arrived at clinically, and must be supplemented by full microscopic study. This may be accomplished by aspiration biopsy, or from tissue removed, as in the case presented. The writer suggests that aspiration biopsy should be attempted, as it is considered less hazardous than operative procedure for biopsy. Various other tumors such as lipoma, submaxillary salivary gland tumor, cervical lymphatic node metastasis, lymphosarcoma, neurogenic sarcoma, cystic hygroma, and carotid body tumor, must be ruled out. There is a marked similarity of two growths originating in the carotid bifurcation, schwannoma and carotid body tumor, both growths being exceedingly rare. Neither can be diagnosed clinically, although pulsation of the carotid artery over the tumor itself has recently been considered an outstanding characteristic of carotid body tumor. The schwannoma has no such clinical characteristic sign of dependable diagnostic value, although under the microscope characteristic histologic factors are recognizable as mentioned above.

In common with all tumors of nerve origin, these schwannomas are markedly radio-resistant, and radiation is contraindicated. Sufficiently small doses of radiation to be harmless are ineffectual, and intensified treatment may cause damage to adjacent tissues.

Surgical removal may be resorted to as means of relieving mechanical pressure due to the growth, or for psychological or cosmetic reasons where the size of the growth has become sufficient to produce disfigurement. It does not metastasize, and from experience in surgical excision no recurrence is reported. However, where the mass is still small and the patient is suffering no marked discomfort, but is apprehensive regarding surgery, removal may be deferred indefinitely without risk, as progress of the growth will undoubtedly be slow and threat of malignancy unlikely.

REPORT OF A CASE

The case to be presented in this report is that of a young Caucasian male, 20 years of age when first seen in March 1944. His chief complaint at that time was some slight increase in irritation of the throat, more noticeable since his recovery from measles two weeks previously.

Fig. 1—Tumor After Removal.

A tonsillectomy at the age of eight years revealed some irregularity of the throat, which was attributed to an after-effect of diphtheria contracted 18 months earlier. In 1940 an ophthalmic examination brought out some abnormality in function of the left eye. There was slight indication of Horner's syndrome noted about this time. In 1941, in a routine check-up, the throat irregularity was again reported, and the patient was advised to watch the progress. He states he was told he had a "fleshy tumor" in his throat, though this did not cause him much discomfort at the time. However, he became somewhat more throat-conscious as time went on. He was also aware of a developing variation in his eyes, in that the left eye was set more deeply in the socket than the right. Being gradual in development, this condition was one to which he had become accustomed, with a consequent degree of adjustment to the syndrome.

On the first visit, the throat examination revealed submucosal swelling on the left side extending from the level of the palate down to the hypopharynx which appeared to obstruct the pyriform sinus. The tumor appeared to be hard and movable with the muscles of the neck. The patient stated that the tumor had existed for about three years. X-ray examination of the neck and clinical examination were inconclusive.

A specimen from the throat consisted of a piece of soft, grayish white tissue, measuring 6 mms. in greatest dimension. Microscopic examination of the tissue submitted showed it to be made up almost entirely of tumor substance. The tumor had a background of pale eosin-staining, homogeneous tissue, through which a few

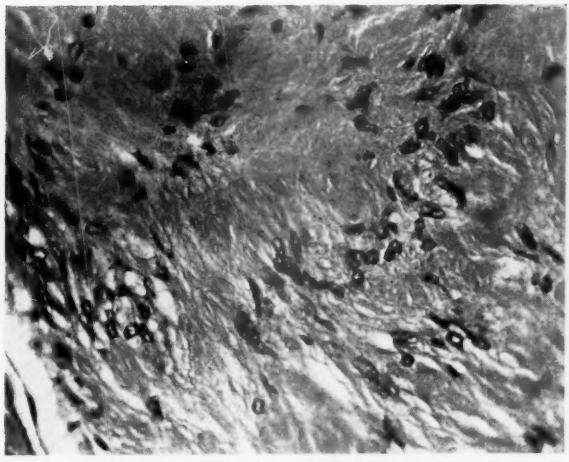


Fig. 2.—Photomicrograph of Section of Tumor. x375.

scattered cells were seen, with spindle-shaped nuclei. These cells were arranged in palisade formations and small bundles. The nuclei were uniform in size and showed no pleomorphism. The histologic picture was typically that of neurofibroma of the schwannoma type.

At operation the mucous membrane was anesthetized with 1% novocaine and adrenalin, which facilitated separation of the membrane from the capsule of the tumor, and also was a factor in producing hemostasis. A vertical incision was made over the prominent portion of the tumor, posterior to the posterior pillar. Blunt dissection of the tumor mass was employed, as well as finger dissection, along the line of cleavage between the soft tissue and the capsular tissue, this being considered a safer procedure than the use of sharp instruments.

The tumor mass extended into the nasopharynx and also into the hypopharynx. The new growth was removed as one mass, and the resulting cavity was found to be lined with smooth, glistening fascia. A very small amount of bleeding was encountered, and the patient made an uneventful recovery.

The patient reported improvement in his ability to swallow when first seen following the operation. Immediately after the

operation the left eye had seemed to be more markedly affected by the Horner's syndrome, but this condition improved very satisfactorily as time progressed. The vision has never been affected at any time, according to the patient's report, and at present is still normal, though the left eye seems somewhat sensitive to light.

The pathological report was as follows: The gross specimen consists of an encapsulated, firm, irregular, nodular mass, of ovoid shape, measuring 75 x 58 x 40 mm. The cut surface is glistening, firm, and grayish-white in color. On it there are irregular whorls and strands.

Histologic examination shows a tumor composed of thin, spindle-shaped cells of connecting-tissue type, which are supported upon a moderately dense fibrillary intercellular stroma. These cells are arranged in irregular pseudo-anastomosing strands, and form palisade-like patterns about central zones of hyaline degeneration. The nuclei are elongated, ovoid in shape, and are of uniform morphology and staining reaction. There is no evidence of malignancy. Diagnosis: Neurofibroma.

SUMMARY

The extreme rarity of schwannoma of the pharynx is indicated by the infrequency of reports in the medical literature. It is generally accepted that this tumor originates in the cells of Schwann, it being a peripheral nerve growth. While bearing many characteristics in common with carotid body tumor, it must be carefully differentiated from this cervical growth, as well as from pharyngeal lymphosarcoma.

Aspiration biopsy is recommended as being less hazardous than operative biopsy. Diagnosis cannot be established clinically; histologic study must be the determining diagnostic procedure.

Horner's syndrome as a concomitant of the tumor, while still very rare, would seem to be increasingly observed, perhaps due to the greater use of present-day neurosurgery, as well as to the increase in traffic and industrial accidents.

Age and sex seem to have no recognizable bearing on the occurrence of this growth.

Radiation is contraindicated, as being ineffectual in small doses, while intensified treatment may prove hazardous to adjacent tissues.

This tumor is a benign growth, invariably slow in developing. It does not metastasize, and no recurrence is reported following excision.

1930 WILSHIRE BOULEVARD.

REFERENCES

1. Ehrlich, Harry E., and Martin, Hayes: Schwannomas (Neurilemomas) in the Head and Neck, Surg., Gyn. & Obst. 76:577, 1943.

2. Figi, F. A.: Solitary Neurofibroma of the Pharynx, Arch. Otolaryng. 17:386, 1933.

3. New, G. B., and Childrey, J. H.: Tumors of the Tonsil and Pharynx, Arch. Otolaryng. 14:596, 1931.

4. Hall, Colby, and Owens, Harold: Solitary Neurofibroma of the Pharynx, Arch. Otolaryng. 34:1163, 1941.

5. Verocay, J.: Multiple Geschwuelste als Systemerkrankung am nervoesen Apparate, Festschrift f. Chiari, Wein und Leipzig, 1908, p. 378.

6. Verocay, J.: Zur Kenntnis der "Neurofibrome", Beitr. path. Anat., 48:1, 1910.

7. Stout, A. P.: Peripheral Manifestations of the Specific Nerve Sheath Tumor (Neurilemoma), Am. J. Cancer 24:751, 1935.

8. Coates, G. M.: Schwannoma of the Mouth, Arch. Otolaryng. 34:1166, 1941.

9. Masson, P.: Experimental and Spontaneous Schwannomas (Peripheral Gliomas), Am. J. Path. 8:367, 1932.

10. Murray, M. G., and Stout, A. P.: Schwann Cell vs. Fibroblast as the Origin of the Specific Nerve Sheath Tumor, Am. J. Path. 16:41, 1940.

11. Murray, M. G., and Stout, A. P.: Demonstration of the Formation of Reticulin by Schwannian Tumor Cells in Vitro, Am. J. Path. 18:585, 1942.

12. Turchik, Frank: Personal communication.

XXX

SOLITARY EXTRAMEDULLARY PLASMOCYTOMA: CASE REPORT

CHARLES E. CONNOR, M.D.

ST. PAUL, MINN.

A 57-year-old man presented himself because of recurring epistaxis of several months' duration, the last attack, on the night before examination, of such severity that he had been unable to continue at his work in a war plant.

Examination revealed a chronic rhinitis and sinusitis of long standing, both allergic and infectious in character, in which the patient was not greatly interested. The posterior tip of the right inferior turbinate was much hypertrophied, the so-called "mulberry tip." A small, globular, sessile tumor about one centimeter in diameter was seen on the upper surface of the right soft palate just anterior to its free margin and close to the lateral pharyngeal wall. The tumor was firmer than a polyp, slightly more flesh-colored, and presented on its posterior aspect (mirror inspection) a small area which appeared as though it might have been the site of recent hemorrhage; the tumor had a definite tendency to bleed on probe palpation.

On the anterior end of the left inferior turbinate was an infiltrated area of mucosa about four millimeters in diameter; this was not ulcerated but also had a tendency to bleed on probe palpation.

Biopsy was made of the palatal tumor; the pathological report was as follows: "There is a small tumor covered by stratified squamous epithelium. It consists of plasma cells, sometimes in solid masses and at other times scattered within hemorrhagic or edematous stroma. Narrow, branching, connective tissue trabeculae course through the tumor".

The diagnosis was plasmocytoma—not malignant.

Accordingly, the palatal tumor was removed surgically and the small area of infiltration on the left inferior turbinate destroyed by electrocoagulation.

Presented at the Continuation Center of Study, Minneapolis, Minn., Jan. 15, 1946.

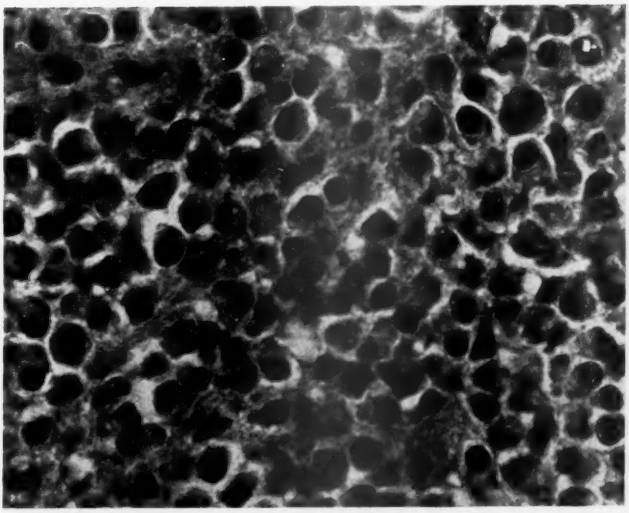


Fig. 1.—Solitary extramedullary plasmocytoma (X 600) showing typical "cartwheel" nucleus, unstained paranuclear vacuole, very little stroma.

On going briefly into the literature of plasmocytoma, differing opinions are found concerning the frequency of its occurrence and its potential malignancy.

It is not a frequent tumor. Hellwig,[1] in a comprehensive review of the literature from 1905 to 1943, found 127 cases and added one of his own. In 64 cases the tumor was in the upper air passages and so is apt to come first to the attention of the otolaryngologist; 47 were in the conjunctiva, 4 in the lymph nodes and 13 in other widely varying locations. Gros,[2] Eagleton[3] and Cappell and Mathers[4] each reported one in the temporal bone. Figi, Broders and Havens[5] reported 11 cases of plasmocytoma in a series of 2885 malignant tumors of the nose, nasopharynx, sinuses, pharynx and larynx examined at the Mayo Clinic; certainly the tumor is a rare, not a common one.

The plasma cell is considered a derivative of the large and small lymphocyte of the blood and of the connective tissue lymphocyte, the fibroblast of the vascular adventitia; the process is one of individual differentiation not accompanied by mitosis and presumably caused by an unknown toxin.

The cell has been reported in ordinary nasal polypi, in mucous and serous membranes, in the blood in cases of multiple myelomata, in the spleen, liver, ribs, sternum and spine in cases of plasma cell leukemia and in the connective tissue of many organs. It may be found, together with its transitional forms, wherever lymphocytes are found, especially in chronic inflammatory processes such as encephalitis, trachoma, tuberculosis and syphilis. The plasma cell is not recognized as a normal constituent of bone marrow.

The histologic structure of the plasma cell is remarkably uniform. The cytoplasm is abundant, non-granular and basophilic, assuming various forms, round, oval, polygonal, due to intercellular pressure. The nucleus is usually single but may be multiple, and is small and eccentric; a paranuclear unstained vacuole may be seen. The chromatin is arranged in several deeply staining blocks about the nucleus, the so-called cartwheel arrangement; characteristic staining reactions are obtained, especially that of Pappenheim; the number of mitotic figures varies greatly.

The stroma is not conspicuous but can be demonstrated by silver staining; it appears as a fine reticulum of hyaline collagen material. The cells are usually packed densely in the reticulum but may be arranged in orderly rows suggesting invasion of pre-existing connective tissue; they may form collars about capillaries and arterioles. Giant cells, eosinophiles, histiocytes, lymphocytes and macrophages are occasionally seen.

There exists no uniformity of opinion as to the exact nature of plasmocytomata or their relation to other members of the group of tumors known as lymphomata; some writers[3] consider them the result of a low-grade avirulent infection which initiates a metaplastic process in bone marrow; others look upon them as granulomatous in origin because of the frequency with which plasma cells as well as lymphocytes are found associated with chronic inflammatory processes. Still others see definite indications of their neoplastic nature in such histologic characteristics as the delicate reticulum, the constant uniformity of cell structure and arrangement, the absence of any considerable number of cell types and the presence of lymphoblasts, promyelocytes and aberrant or giant forms of the plasma cell.

Whether or not solitary plasmocytomata are benign or malignant is a question upon which there is also considerable difference of opinion. Some authors consider them benign, citing patients who remained well for many years after local treatment; however, one

patient with a solitary plasmocytoma is reported who remained well for eight years after local treatment, only to return with multiple myelomata. Case studies such as these, together with the fact that the histology of the tumor gives no indication of its potential malignancy, call for a guarded prognosis in any such case, even though it presents no local evidence of malignancy or generalized metastasis; the presence of these latter findings settles the issue.

The plasmocytomata, composed of cells derived from lymphocytes, belong to the group of tumors known as lymphomata, the members of which are lymphocytomata, locally benign or with a tendency to metastasize, lymphosarcomata, multiple myelomata and lymphatic leukemia. Cases such as that described above suggest that there may be a definite relation between solitary plasmocytomata and malignant multiple myelomata.

Jackson, Parker and Bethea,[6] in reporting a series of plasmocytomata, summarize the situation concisely: "It would seem that there is no very sharp line of demarkation between the localized benign plasmocytomata on one hand and the malignant, fatal multiple myelomata on the other. No clear distinction can be seen, again, between the nodular circumscribed form and the diffuse form".

The plasmocytoma varies greatly in its size and gross appearance. It ranges from a tumor a few millimeters in diameter to a mass filling the nasopharynx or antrum. It may be smooth, nodular, or coarsely granular and in color, pale, yellowish gray, bluish red, or dark brown. It may be pedunculated, sessile, or a diffuse thickening of the mucous membrane; it may be firm, or soft like a mucous polyp. It may present evidence of recent hemorrhage and ulceration or necrosis, especially of bone.

Clinically, the plasmocytomata of the upper air passages are seen as noncancerous single tumors, noncancerous multiple tumors, cancerous tumors without metastasis but with local infiltration or ulceration and cancerous tumors with metastasis in lymph nodes or bone.

The symptoms are those of expanding, eroding tumors similarly situated: swelling, obstruction, headache, pain, difficulty in swallowing, breathing or speaking, deafness and impaired vision.

These tumors, when seen early, are often quite small and confined to mucous membrane. In this stage they may be locally controlled by surgical removal or radiation; after they have metastasized to other structures, especially to lymph glands or bone, there is no

cure. The inherent uncertainty of the prognosis substantiates the advice of Figi, Broders and Havens to consider plasmocytomata malignant and to treat them as such.

1371 Lowry Building.

REFERENCES

1. Hellwig, A. C.: Extramedullary Plasma Cell Tumors as Observed in Various Locations, Arch. Path. 36:95 (July) 1943.

2. Gros, J. C.: Plasmocytoma of Temporal Bone, Arch. Otolaryng. 42:188 (Sept.) 1945.

3. Eagleton, W. P.: A New Classification of the Bones Forming the Skull, Arch. Otolaryng. 24:158 (Aug.) 1936.

4. Cappell, D. F., and Mathers, R. P.: Plasmocytoma of the Petrous Temporal Bone and Base of Skull, J. Laryng. & Otol. 50:340 (May) 1935.

5. Figi, F. A., Broders, A. C., and Havens, F.: Plasma-Cell Tumors of the Upper Part of the Respiratory Tract, Annals of Otology, Rhinology and Laryngology 54:283 (June) 1945.

6. Jackson, Henry, Jr., Parker, Frederic, Jr., and Bethea, J. M.: Studies of Diseases of the Lymphoid and Myeloid Tissues. II. Plasmocytomata and their Relation to Multiple Myelomata, Ann. Int. Med. 181:169 (Feb.) 1931.

XXXI

AEROTITIS MEDIA IN SUBMARINERS

HENRY L. HAINES, M.D.

J. DONALD HARRIS, Ph.D.

NEW LONDON, CONN.

Aerotitis media is characterized by middle ear lesions caused by failure to equalize pressure between the middle ear and the surrounding atmosphere. The pathological changes vary from slight congestion to extensive rupture of tissues. Discomfort and pain are usually present; there may be tinnitus, vertigo, and deafness, and in the severer cases, bleeding. Aerotitis media is also known as aero-otitis, aerotitis, otitic barotrauma, otic barotrauma, salpingotympanitis, and aviator's ear, among other terms.

In the past few years aerotitis media has become a serious problem with the increase of high altitude flying and of diving and salvage operations and with the large expansion of the Submarine Service. It is now recognized as an occupational injury, both in and out of the Armed Forces.

Because of the incomplete and often confusing statements which have been made on various aspects of the disorder, in our work we felt a necessity for especial care in experimental design and in application of control procedures. Merely another clinical study would not meet the need. In view of the fact that we had a rich source of material, and since every condition necessary for rigid experimental control was available, we felt it possible to meet rigorous research criteria, and thus to provide a fairly definitive study on the causes, effects, prediction, and treatment of the disorder. The present report describes aerotitis media as it appears in submariners, and recounts the experiments by which we attempted to understand it and to mitigate its effects.

Before World War I, only sporadic accounts of ear damage under conditions of changing pressure appeared. There are antique accounts of disorders in pearl divers of the Orient, and of ballooners even before the Wrights. Beaven[2] and Campbell[1] have given much of the early history, mentioning a case of aerotitis media described by Pilatre de Rozier in 1783 after descent from a balloon journey,

and giving Glarsher and Coxwell's description of the symptoms in 1862.

Shilling and Everley[18] have recounted the history of aerotitis media particularly as seen in divers, salvage workers, caisson workers, and submariners. Requarth[15] contributed to the literature on pressure effects on caisson workers, particularly the treatment of caisson disease with helium and oxygen mixtures.

Accelerated research on aerotitis media has kept pace with its growing seriousness as a result of the great increase in high altitude flight during World War II. In 1941 Poppin[13] considered aerotitis media as seen in flying personnel. The use of radon in the treatment of aerotitis media began with Fowler,[8] who treated successfully many cases in the Eighth Air Force by the Crowe and Burnam technique.[6] The United States Army Air Forces soon thereafter launched a major attack on the problem, of which a report will shortly appear.

The work of the present paper stems from the investigations of Shilling and Everley,[18] who saw that the air forces would experience difficulty with aerotitis media, and from the later investigations of Teed,[20] who discussed the factors producing obstruction of the eustachian tubes in submariners.

Preliminary accounts of our work have already appeared.[9, 10, 17]

Inasmuch as it is an abnormal functioning of the eustachian tube which is the inciting cause of aerotitis media, its anatomy should be described. The tube consists of a bony and a cartilaginous part. The bony part is roughly a flattened funnel in shape, is about 12 mm. long, and arises from the upper part of the anterior wall of the tympanic cavity. Its mucous membrane, which lies close to the bone, is covered with ciliated epithelium. The bony part passes downward, forward, and medially, and joins the cartilaginous portion without a definite line of demarcation. The juncture is known as the isthmus, and is the narrowest portion of the tube.

The cartilaginous portion is about 24 mm. long from the isthmus to its opening high up on the lateral wall of the nasopharynx. Its size increases as the pharynx is approached, but normally its walls are apposed and it remains a vertical slit-like potential tube unless opened by action of voluntary muscles or forced open by positive pressure in the middle ear. It is lined with columnar ciliated epithelium on a loose stroma containing mucous glands and diffuse lymphoid tissue.[7] The cilia create a current toward the nasopharynx thus helping to drain the middle ear. The lymphoid tissue near the

mouth of the tube is known as the tubal, or Gerlach's tonsil. This should not be confounded with the lymphoid tissue in the fossa of Rosenmueller, the part of the nasopharynx behind the torus tubarius of the pharyngeal opening of the eustachian tube.

The end of the tube, the pharyngeal ostium, acts as a flutter valve in that as a result of its contour it opens easily to allow the escape of air under positive pressure in the middle ear, and yet unless opened voluntarily it prevents passage of air into the middle ear.

Of the muscles near the eustachian tube, the tensor veli palatini is the most important in opening the lumen.[16]

The eustachian tube is normally closed, entrapping the air in the middle ear; it follows, that with a change in ambient air pressure there results a pressure difference across the tympanic membrane.

If the ambient air pressure is decreasing, the relative pressure of the air in the middle ear builds up until it is about 15 mm. of mercury greater than that of the air in the nasopharynx, at which time the eustachian tube is forced open and enough air escapes to equalize the pressure. As the ambient air pressure continues to decrease, the process is repeated.

On the other hand, if the ambient air pressure is increasing, the mechanical effect of the pressure difference is not exerted to open the tube—rather it is exerted to keep the tube closed. The eustachian tube must then be opened by action of its muscles. A sense of fullness develops in the ears at a pressure difference of 3-5 mm.* Discomfort is noted if the pressure is allowed to increase to 15-30 mm. Here there is diminished hearing acuity as a result of retraction of the tympanic membrane and fenestra ovalis and consequent telescoping of the ossicles. Above 30 mm. differential pressure there may be pain, tinnitus, bleeding, and even vertigo and nausea. Pressure differences of 80-90 mm. give great pain and increase the severity of all symptoms.

A difference of 80-90 mm. is usually above that which the tubal muscles can overcome, whereupon with any increase in pressure the tube becomes "locked"—the walls are pressed so tightly that the muscular reflexes, swallowing, shouting, forced expiration while holding the nose (the Valsalva maneuver), which normally open the tube are unable to exert their effect. The outside pressure must then be reduced before equalization can occur.

---

*These figures are all for sea level air density.

Many persons are not able to inflate the middle ear voluntarily. There may be colds, with infection and swelling of the lymphoid tissue underlying the epithelium of the eustachian tube so that the lumen is entirely closed, or there may be sufficient excess tissue to block the tube even with no infection. There may be other types of congestion, or there may be paralysis or atypical functioning of the tubal dilator muscles.

But even with individuals who can ordinarily inflate the middle ear voluntarily, it may occasionally not be done, as during sleep (babies in airplanes are particularly susceptible), unconsciousness, or during the condition of "locked" tube. In addition, a person may be able to equalize pressure at one rate of pressure change but not if the rate be increased.

With an individual unable to inflate the tympanum, or unable to do so with sufficient speed, a series of pathological events will occur in the tissues of the middle ear whenever positive pressure is encountered. These events are characterized by vascular rupture and by the separating of tissue layers.

The immediate cause of the otopathy appears to lie in a pressure differential between the entrapped air in the tympanum, and a component of ambient air pressure transmitted to and expressed in the tissues involved. When this occurs, so that for example within a capillary or arteriole the pressure is greater than in the air of the tympanum, the vessel will expand and may rupture. The same "suction" effect may cause the outer layers of some tissues to be pulled loose.

We have followed Teed[20] in grading the severity of symptoms. No. 0 is a perfectly normal ear. A No. 1 ear shows some congestion in Shrapnell's membrane and along the manubrium. A No. 2 ear shows fiery red congestion of the entire tympanic membrane; a No. 3 ear has in addition evidence of ruptured vessels in the tympanic membrane; a No. 4 ear has extensive vascular rupture, with free bleeding in the middle ear and from the eustachian tube. There may be dissecting hemorrhages in the layers of the tympanic membrane, the membrane may actually be ruptured, or there may be bleb formation in the canal. A No. 5 ear is one in which the whole middle ear is filled with blood and serum unmixed with air, the tympanic membrane appearing purple or black—due to lack of aeration.

A few patients develop vertigo and nausea. Commonly, pain occurs radiating down the side of the face to the throat, or it may be deep-seated in the ear. Sometimes the pain is rather mild; a

tympanic membrane may rupture before the patient feels more than a transitory needle-like pain. Often there is tinnitus, usually a roaring noise. Stuffiness and "dullness" of the ears is common. Many complain of reduced auditory acuity, though we have found the subjective feeling of deafness usually to be an artifact.

EXPERIMENTAL DESIGN

1. *The Administration of Pressure.* The Submarine Escape Training Tank is a tower containing a column of clear fresh water 25 feet in diameter and 100 feet high. Escape trunks are located at depths of 18, 50, and 100 feet, constructed in a manner similar to the escape trunk of a submarine. Men are required to enter a trunk, submit to a pressure increase appropriate to the depth of the trunk, don an oxygen-supplying and CO_2-absorbing mask, pass from the trunk to the water, and ascend to the surface.

As a preliminary check on the ability of the men to undergo the pressures involved in these trunk escapes, they are first required to enter a dry recompression chamber where they are subjected to 50 pounds (3.4 Atmospheres) pressure in from 3 to 10 minutes. Fifty pounds is as much as the men will meet in the course of their escape training, and it is with the dry recompression phase only that this experiment deals.

2. *Subjects.* All subjects were young healthy men of normal or above normal intelligence, who had passed on the preceding day a very rigorous physical and psychiatric examination for entrance to the Submarine Service. They were all volunteers, with a relatively high state of motivation to do well under pressure.

3. *Prepressure Examination.* On the day before the pressure test, each man received a pure tone audiometer test at the six octaves, 256 to 8192 cycles per second, with a Western Electric 6B audiometer in a thoroughly soundproof anechoic chamber. Each man was examined with the otoscope and the nasopharyngoscope, the condition of his whole auditory system being recorded in detail. Condition of the tympanic membrane and the adenoids, were graded on objective scales. Any unusual conditions were noted. Those who had severe colds or who for some other reason it was thought would be unable to withstand pressure were excused until their troubles were corrected. There were, for example, a few men with psychological disturbances arising from the imminence of pressure. When all men had been thoroughly examined, they were sent for a pressure test.

4. *Postpressure Examination.* Immediately after pressure each man was examined carefully with the otoscope. Every ear was assigned a grade of damage according to the graded scale. A postpressure audiogram was taken. At the sign of any pathological changes in an ear, that man was examined with the nasopharyngoscope. Daily protocols were kept to facilitate pre- and postpressure comparisons.

5. *Experimental and Major Control Groups.* Of those not contracting aerotitis media, a group was required after the lapse of a week to take pressure a second time. In this as with all other groups, the per cent of those contracting aerotitis media and the average grade of ear damage were noted and made the basis for group-to-group comparisons.

Of those contracting aerotitis media, one control group was required after the lapse of a week to take pressure a second time, no treatment whatsoever being given in the interim. Other patients were assigned at random to experimental groups for treatment as follows:

a. Psychological Treatment. A man was assured that in 10 days or less his ears would heal and he would experience no difficulty with a second pressure test. He was subsequently instructed again in the Valsalva maneuver and given a second pressure test with the otologist present in the chamber for individual example and instruction.

With separate groups, the effects of music and of chewing gum were studied.

b. Symptomatic Treatment. Every two hours for six to eight hours before a second pressure test, patients were instructed to apply drops of .25% neosynephrine in physiologic sodium chloride solution to the nose.

c. X-ray Treatment. Our program for investigating x-ray as a means of shrinking excess tissue around the pharyngeal ostium was discontinued for administrative reasons before conclusive results were obtained. Although we do not have data of our own we feel that x-ray treatment should prove beneficial.[14]

d. Dental Treatment. In collaboration with the present writers, a dentist, selected a number of men unable to take pressure whose dental occlusion was not normal. It was reasoned that dysfunction of the temporomandibular joint could affect the normal operation of the eustachian tube. Remarkable results were achieved, and we believe

that dental treatment is definitely indicated in certain cases. A complete description of the technique and results appears elsewhere.[11]

e. Radium Treatment. A random sample was selected from those patients with excessive lymphoid tissue in and about the pharyngeal ostium. Fifty milligrams of radium salt was applied for 8 to 10 minutes. The radium was contained in a monel metal applicator 2 cm. long, inside diameter 1.7 mm. The walls were .3 mm. thick. This cylinder was brazed to a wire by which it was handled. We followed Crowe's and Burnam's technique[6] except for the omission of local anesthetic and astringents. The patient is placed on his back, the applicator passed along the floor of the nose until the middle of the cylinder touches the orifice of the eustachian tube. The applicator is not inserted into the tube. The correct depth can be determined by inserting a nasopharyngoscope in one side and a calibrated dummy applicator in the other, observing the dummy applicator with the nasopharyngoscope.

A similar applicator is then placed in contact with the orifice of the other eustachian tube. A clamp may be placed over the handles of the applicators in such a way that the radium is forced outward against the tissues it is desired to shrink.

Successive radium treatments were given to individuals at intervals of about a month. One group was required to take a pressure test after every treatment, while another group was required to wait until a course of three to eight treatments was completed.

RESULTS

1. *Volume of Work.* A total of 6,149 men were examined before and after pressure during the months of this study. Of these, 26.9 per cent contracted aerotitis media. A total of 732 patients were given radium treatment, 50 patients were given dental treatment, 264 were given symptomatic treatment, and over 200 were given psychological treatment. Some patients with minor symptoms were given no treatment at all.

2. *The Effect of Weather.* It has often been supposed that weather conditions influence the incidence of aerotitis media, but we conclude from our data that their influence, if any, is indirect. Inspection of day-by-day figures for temperature, barometric pressure, and incidence of aerotitis media does not reveal any tendency for the disorder to be associated with these meteorological conditions.

Neither is there any tendency for a higher incidence at certain seasons. In this we do not confirm Teed, who found winter months

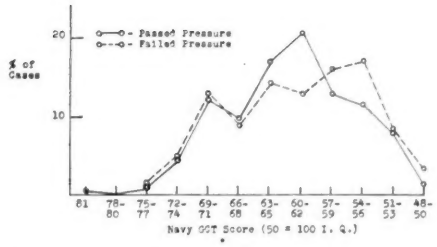


Fig. 1.—Relation of intelligence to ability to pass pressure test.

produced more trouble. It may be that our strict exclusion from pressure of men with pronounced colds reduced the slight seasonal effect which Teed found. As we shall show, however, the presence of colds is of less significance in aerotitis media than is usually claimed.

3. *The Effect of Intelligence.* Figure 1 presents a graph of intelligence of a group of men who passed pressure, and one of a group which failed. A tendency can be detected for the curve of those who failed to be shifted to the right hand or "low" side of the intelligence continuum. However, the difference between the averages is statistically insignificant.

It would be the part of caution not to assume that intelligence is unrelated to the problem. Our men are highly selected. We can say nothing of the behavior under pressure of men with lower grades of intelligence, but it is possible that with a wider range the factor of intelligence would take on added significance.

4. *Prediction of Aerotitis Media.* a. Objective Colds and Allergic Upper Respiratory Manifestations. Because of the prevalent opinion that upper respiratory infections contribute heavily to the onset of aerotitis media, we investigated whether most of the patients gave a history of a cold at the time of the pressure test. In one sample of 232 patients 83.7 per cent had no colds whatsoever, and we conclude that colds or allergic manifestations are by no means always an accompaniment of aerotitis media. At least within the limits of our data the relation of mild colds to aerotitis media is not very pronounced.

b. Size of Adenoids. In view of the close approximation of the adenoids to the pharyngeal ostium and their effect on the function

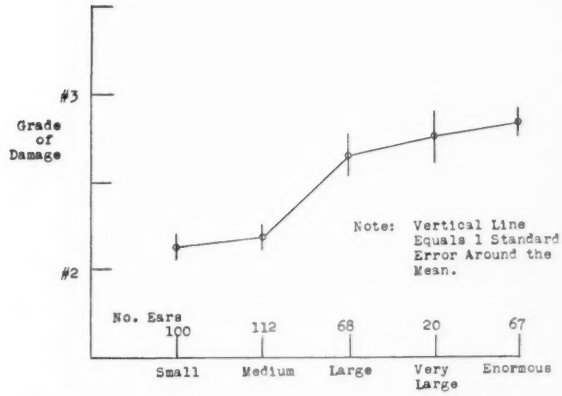


Fig. 2.—Ear damage as related to size of adenoids.

of the latter, one would expect that with larger and larger adenoids, more and more aerotitis media would occur. That this is in general true is concluded from Fig. 2. where the average grade of ear damage is related to size of adenoids. There is a strong and reliable tendency for the damage to increase in severity as the size of adenoids increases.

A sharp rise in Fig. 2. occurs as the adenoid size increases from "medium" to "large". Almost certainly the explanation lies in the fact that the "large" category includes those cases with lymphoid tissue in the fossa of Rosenmueller, while the "medium" category does not. It seems that the fossa of Rosenmueller occupies a strategic position with regard to its effect on the patency of the eustachian tube.

But the relationship between size of adenoids and severity of aerotitis media is not nearly so precise as appears from the condensed data in Fig. 2. A great deal of overlap occurs such that many patients with enormous adenoids have no trouble whatsoever with pressure, while on the other hand many with small or no adenoids sustain severe damage. Fig. 3 presents frequency distributions of each of four groups, those with small, medium, large, and enormous adenoids. Whereas 50 per cent of the "enormous" group does contract aerotitis media, nevertheless over 20 per cent of the "small" group does also—a sizable proportion. In addition it will be noted

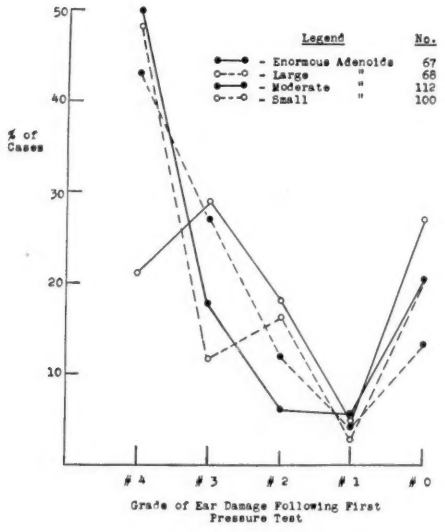


Fig. 3.—Shows lack of precise relationship between size of adenoids and severity of aerotitis media.

that about 20 per cent of the "enormous" group maintain perfectly normal ears.

c. Condition of Eustachian Tube. It was hoped that the appearance of the eustachian tube as seen by the nasopharyngoscope without astringents* might prove a datum that would lead to successful prediction. Fig. 4 shows the relation of the condition of the tube to the grade of ear damage. A regular increase in severity of damage occurs as the eustachian tube becomes more and more occluded.

For purposes of prediction, however, the regular and statistically reliable rise in the curve of Fig. 4 is misleading. The overlap in Fig. 5, drawn on the same coordinates as Fig. 3, shows that the severer grades of ear damage are by no means confined to those patients with covered or closed tubes—in fact more than 25 per cent of patients with perfectly open tubes suffered No. 4 ears, while conversely about 15 per cent with covered tubes had no trouble whatsoever.

*Some of the differences between our observations and those of the Air Forces may be due to the fact that we never cocainize the tissues or use any astringent.

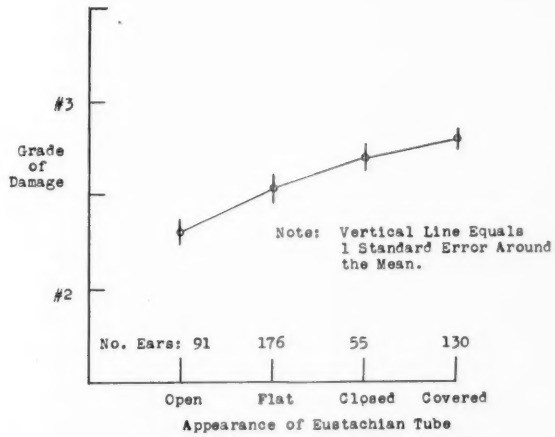


Fig. 4.—Ear damage as related to condition of eustachian tube.

We conclude that a definite relation exists between the appearance of the eustachian tube and ability to take pressure, but that the individual variation is too great to allow accurate prediction of just which men will contract aerotitis media.

d. Ability to Perform the Valsalva Maneuver. In addition to studying the static appearance of the system, we wished to obtain an idea of its functional properties as well, in the form of the Valsalva maneuver. Before the pressure test, a long series of men was checked on how well they could voluntarily inflate the middle ear. The otologist watched the tympanic membrane with the otoscope while the man attempted the maneuver.

The records of 139 men who subsequently contracted aerotitis media were studied. It was found that 56 per cent apparently had had no difficulty with inflation beforehand; 31 per cent were listed as having had some difficulty beforehand, while the remaining 13 per cent were in a "poor" category. Evidently the ability to perform the Valsalva maneuver during a preliminary examination is no certain guarantee that during actual pressure the maneuver will always be successful.

We may specifically inquire what the chances are of a man contracting aerotitis media if he is labelled good, doubtful, or poor on a preliminary test of the Valsalva maneuver. Table I gives the answer. For this table a total of 588 men who did not contract aero-

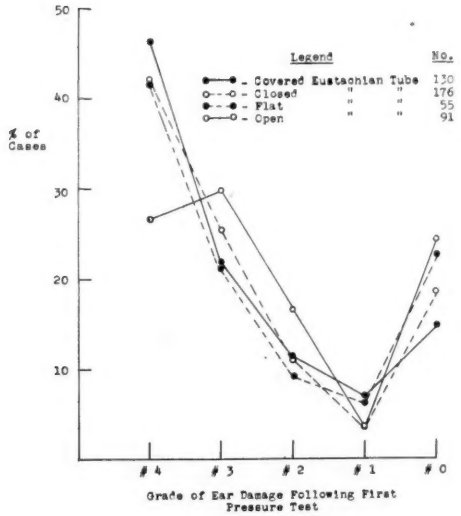


Fig. 5.—Shows lack of precise relationship between appearance of eustachian tube and severity of aerotitis media.

titis media was drawn from the same weekly intervals as the 139 men of the preceding paragraph. Here is shown the prediction of which a preliminary Valsalva maneuver test is capable. Of those labelled "good", only 14.5 per cent contracted aerotitis media. This result at first looks very satisfactory, until it is noticed that the figures are only 11 per cent different for the "doubtful" category. The difference between the figures of 14.5 per cent and 25.5 per cent is reliable at a satisfactory level of confidence (2 per cent level), but it is clear that since about three out of four of those labelled "doubtful" do not contract aerotitis media, it is impractical to prevent these men from attempting the pressure test.

TABLE 1.

SUCCESSFUL PREDICTION OF AEROTITIS MEDIA FROM ABILITY AT THE VALSALVA MANEUVER

	ABILITY AT VALSALVA MANEUVER		
	GOOD	DOUBTFUL	POOR
Total Cases	538	168	21
Contracting	14.5%	25.5%	85.7%
Not Contracting	85.5%	74.5%	14.3%

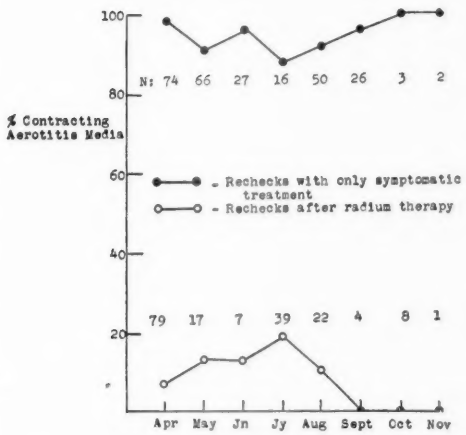


Fig. 6.—Effect of radium therapy on incidence of aerotitis media.

There are perhaps too few cases in the "poor" category to make any final statement, but it is certainly significant that 18 of the 21 men did contract the disorder. It would probably be efficient to prevent all poor cases from taking pressure until they received appropriate treatment.

We conclude that unless a man is in the lowest 2 or 3 per cent, a preliminary test of ability at the Valsalva maneuver provides only a rough guide to a man's ability to take pressure.

e. Previous Experience with Pressure. Repeated exposure to pressure will not of itself result in ability to overcome an initial failure. The top curve of Fig. 6 shows that when patients are given a second test with no treatment or only topical treatment, more than 90 per cent will again contract the condition. We have a number of cases in our files of men sent through pressure a half-dozen times or more, each time waiting until the previous trouble had cleared up; yet each time the same symptoms appeared.

We conclude that if a man can pass our pressure test once he can pass it twice, but if he cannot pass it the first time, he needs more than cursory attention before he attempts it again.

f. Discussion and Conclusions of Prediction Experiments. Our study has failed to find a means of stating with precision whether a

man can sustain pressure without ear damage. We have been disappointed not only in our attempt to predict individual cases, but even in an attempt to lower substantially the overall incidence. We saw from Table I that even if we rejected all who were doubtful and poor at the Valsalva maneuver there would still be 14.5 per cent in the good category who would contract aerotitis media, and we therefore feel that Teed's ideal figure of two to three per cent incidence is not attainable with the use of the Valsalva maneuver alone. It would seem that an incidence of about 15 per cent is a sort of theoretical "floor" below which present selection methods cannot go. At least, in all the thousands of men examined here, our lowest incidence for a reasonably large sample of experienced men has never been lower than 18 per cent.

In this paper we have shown that a sizable percentage of men with small adenoids, open eustachian tubes, and with good ability at Valsalva, still may contract aerotitis media. One reason why this may be so is that, in the pressure chamber, some men may let the increasing pressure "get ahead" of them one or more times, and inflate their ears only after some damage has been done. This may occur even with men perfectly capable of auto-inflation.

Another explanation may be that some men are especially susceptible to pressure, so that even though they are inflating their ears regularly and without discomfort during increasing pressure, nevertheless the slight pressure differential existing each time before the ears are voluntarily inflated is enough cumulatively to cause some otopathy.

5. *Therapy.* a. The Effect of Radium Therapy. Fig. 6 shows that radium therapy has a satisfactory effect. The lower line represents the incidence of aerotitis media in patients after they have completed a course of radium treatments A striking difference is seen between this group and a control group given no radium.

We conclude that radium is effective in about 9 out of 10 men. For those men whom radium does not help, it may be that more radium would be effective, or it may well be that other conditions besides excessive tissue produced the trouble in the first place.

During the final month of our study, for example, three patients failed to respond to radium treatment. In two of these cases old mastoiditis had caused considerable scarring, while the other had a post-diphtheritic paralysis of the right side of the throat and palate. The latter patient failed to move or open the eustachian tube on swal-

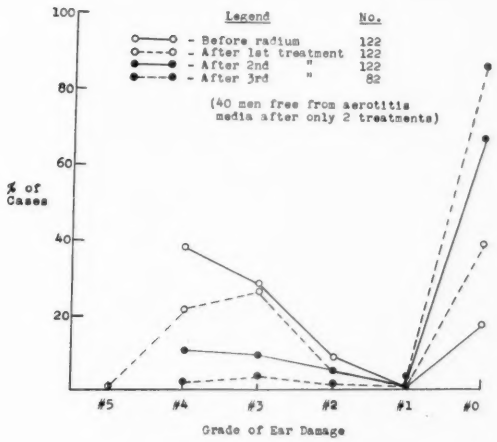


Fig. 7.—Frequency distributions of ear damage following successive radium treatments.

lowing or inflation. His tympanum could, however, be inflated easily with a eustachian catheter. The tympani of the other two men could not be inflated by catheterization.

In order to study the effects of successive radium treatments, a group of 122 men was given pressure after every treatment instead of waiting until the radium series was completed. Fig. 7 gives the percentage of each grade of ear damage before treatment, and the percentage of each grade of damage after each treatment. The graph may be read vertically, thus: 38 per cent of the ears were rated No. 4 before treatment, 21 per cent were rated No. 4 after one treatment, 9 per cent after two treatments, and 2 per cent after three treatments. Conversely, 20 per cent of the ears were rated No. 0 before treatment, and 88 per cent after three treatments.

In these patients, some No. 0 ears before treatment are found because the disorder is not always bilateral; in one series of 250 patients there were 152 bilateral cases, and 55 and 43 with trouble only in the right and left ear respectively.

The average grade of damage for the 122 men throughout the series of radium treatments is shown in Fig. 8. A steady and reliable decrease in damage is seen, declining from nearly a No. 3 ear almost

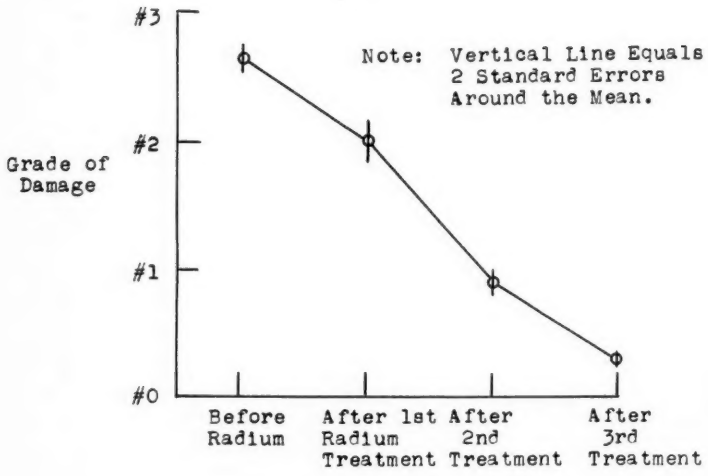


Fig. 8.—Ear damage as related to number of radium treatments.

to a No. 0 after three treatments. Forty men were held for only two treatments, but it is clear from Fig. 8 that most ears sustain little or no damage when subjected to pressure after two radium treatments.

b. The Effects of Topical Therapy. A slight beneficial effect of neosynephrine nose drops was ascertained. The average incidence of aerotitis media on a second pressure test was about 84 per cent. This figure is somewhat better than that with patients receiving no treatment at all.

c. The Effects of Psychological Treatment. A number of procedures of a minor nature were tried. A group of 120 men was instructed to chew gum vigorously during pressure, and compared with a group of 142 men given no gum. Both groups took pressure simultaneously. No difference was found. Music was presented to 276 men as compared with no music to 586 men during the same weeks. No difference between groups was found.

For two months, certain patients were told that their trouble would clear up in a week or ten days, and that they would experience no further trouble. No therapy other than this suggestive sort was given. In addition, they were told that if they did not pass the

second test their off-the-base liberty would be cancelled for a day. Then, during the second pressure test, the otologist took pressure along with the men, encouraging and instructing them individually.

No effect whatever was found from our most strenuous efforts in the psychological direction.

d. Discussion and Conclusions on Therapy. We conclude that since several measures of a psychological nature have been given a fair trial and proved valueless, it is probable that no such therapy will prove effective.

From the very slight improvement with topical therapy, we conclude that effective therapy must necessarily consist of some radical alteration of the structure of the ear system. Our experiments with x-ray were incomplete, but dental treatment, where indicated, and more generally treatment with radium, both produce an extensive change, either the removal of hyperplastic lymphoid tissue around and in the opening of the eustachian tube, reduction in congestion about the tube, or realignment of muscles attached to the tube. It appears that a mechanical alteration must take place if a patient is to sustain pressure successfully in the future.

Treatment of the acute symptoms of aerotitis media is very simple, consisting usually of letting well enough alone. More secondary infections result from attempts to clean out the ear than if it is left strictly without treatment. Extravasated blood will shortly be resorbed, and separated layers of tissue will usually heal in a few days. Very little cicatrization is apparent. To afford some relief, nose drops may be administered or local shrinkage of tissue about the tube with cocaine may be indicated.

6. *The Effect on the Absolute Threshold.* a. History. The literature contains a variety of seemingly contradictory statements on the factor of auditory acuity. It is claimed for example[19] that typically the disorder is characterized by a high tone loss, and[12] that low tones are first and typically affected; again, it is stated[19] that the deafness may be severe and permanent, and[10] that any acuity loss is usually regained in a matter of hours.

There is no question but that most of the apparent contradiction can be explained on the basis of the quite different conditions to which the patients of the several studies were subjected. We now know enough of the relation between aerotitis media and hearing acuity to resolve most of the difficulties, and to approach something like a final statement.

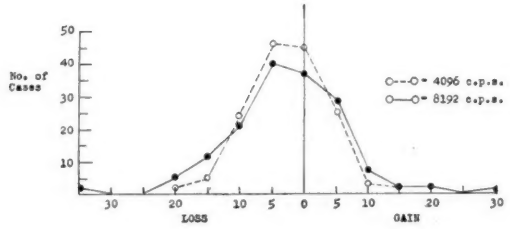


Fig. 9.—Distribution of audiogram changes of 154 men with no pain.

In the early literature on the subject, mostly from studies on caisson workers, the question of the etiology of deafness is complicated by many factors. In the first place, only relatively crude tests of hearing could be administered. In one study[3] on 13 caisson workers, "the hearing tests were made oftentimes under unfavorable circumstances, being made at times in saloons, in boarding houses, and in other places where it was not possible to secure quiet for tests with the whispered voice."

In the second place, the reason for deafness could usually not be determined; it could have been caused by the immediate action of compressed air on the auditory mechanism, by lesions due to previous attacks of aerotitis media, by nitrogen bubbles in the fluid of the inner ear, or indeed may have existed prior to the investigation.

In 1942 Shilling and Everley[18] presented post-pressure audiograms for 18 cases of severe aerotitis media, showing for some subjects a disabling loss in hearing, and describing the course of recovery of hearing. Some of these men had had many years of submarine experience, and their pre-pressure audiograms may have been depressed as a result of gunfire, blast, disease, or the like.

Teed[20] noted that men now and again said they had a subjective sensation of lowered hearing following pressure. Audiometry was not provided. It is our experience, however, that a feeling of fullness or congestion may readily be confused with true lowered acuity. We have seen many scores of men who state that their hearing was "fuzzy" whose audiograms were nevertheless perfectly normal.

Because earlier studies had not controlled conditions so that clear relationships appeared, a thorough-going experiment was needed in-

volving the application of pressure to a large number of normal ears, with complete audiometry both before and after. In our experiment the men had little or no previous otopathy, compressed-air illness could not arise, auditory fatigue could not occur, and in short no complicating factor intruded to prevent determining in clear-cut fashion the effect of aerotitis media on acuity.

b. The Factor of Pain. Teed mentioned the possibility that the degree of pain might be related to severity of damage. This relationship does not hold at least so far as acuity is concerned. Of a group of 183 patients, the audiogram change was no different between those reporting pain and those not reporting pain. On the other hand, it is not necessarily true that those with no pain suffered no loss in acuity. When the difference audiograms at the high frequencies are inspected it can be seen that some loss occurred. Fig. 9 presents distributions for the difference audiograms at the two highest frequencies. The zero vertical represents no difference between the pre- and postpressure audiograms. Since it is clear that there is a preponderance of cases to the left or "loss" side of the zero, we conclude that some loss of acuity may be present even where conditions are favorable and with patients whose discomfort could have been only mild. (Even though these losses are small, they are nevertheless several times their own Standard Errors.)

Haines[9] has mentioned that not all otopathy is accompanied by pain. The extent of this overlap can be estimated from the fact that although more than 25 per cent develop otopathy, only 12 per cent of all ears pain so that men will report it.

It is of interest to examine the differential effect of the damage giving rise to pain. Fig. 10 is constructed exactly as Fig. 9 except that each frequency is given its own coordinates. It is clear that low tones may or may not have been affected, but that for frequencies of 2048 c.p.s. and above, some loss of acuity was usual.

c. The Effect of Rupturing the Tympanic Membrane. The question arose whether rupturing the tympanic membrane lowers acuity. It seems that some lowering occurred in uncomplicated perforations, but it was mild at worst. The average losses of 5 to 10 db. did not conceal a single case where a loss greater than 15 db. had been sustained.

d. The Factor of Grade of Damage. The less severe grades of damage were not accompanied by enough loss of acuity to distinguish from normal ears—indeed in the vast majority of Nos. 1, 2, and 3 ears no loss whatever was found.

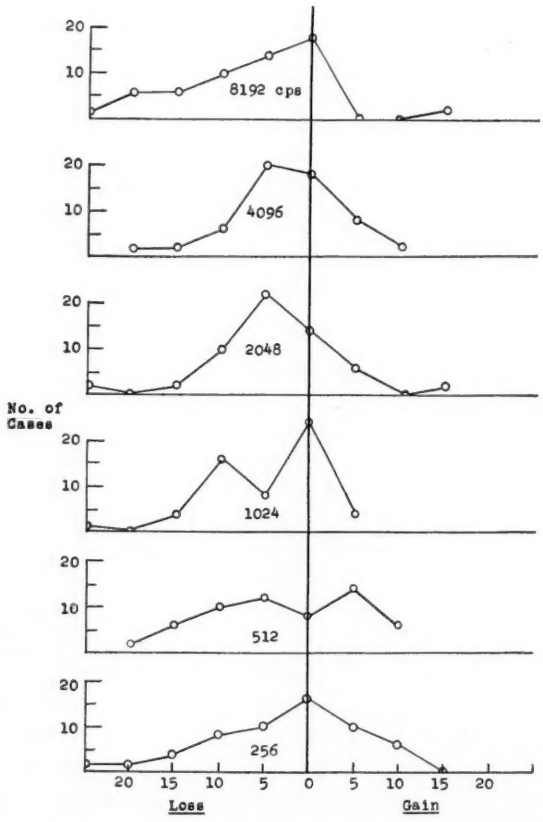


Fig. 10.—Distributions of audiogram changes of 29 men reporting pain.

A composite audiogram for No. 4 ears has been presented.[10] The loss was significant both in the statistical sense and in the sense that the patients were aware of lowered hearing. However, by comparison with the decibel loss we see in those individuals even mildly hard of hearing, the loss in the No. 4 ears was certainly not severe.

As a matter of fact, there were many in this series of No. 4 ears where a loss of acuity was definitely not present. Indeed there were several cases in which at least at some frequencies an improvement in acuity was exhibited. It is apparent that the appearance

of the ear as seen with the otoscope is not a good index to the functional efficiency of the ear.

e. The No. 5 Category. It was necessary to explain in some other way those cases in which a severe loss of acuity did occur. From a more careful perusal of the protocols of No. 4 ears, we noted that the severely affected ears were typically characterized by a dark purple or bluish discoloration of the tympanic membrane, indicating a middle ear filled with dark blood unmixed with air. We found this to be the case for all of the severely deaf ears and for none of the other No. 4 ears. Accordingly we distinguished the condition as No. 5. Fig. 11 represents the loss in acuity for the No. 5 ears. It must be emphasized that these ears do not show any greater amount of pathological changes than No. 4 ears—the essential difference is merely the presence in the middle ear of free serosanguineous fluid without any entrapped air.

It is our hypothesis that the severe deafness occasionally encountered in aerotitis media among submariners is not caused by insult to the tissues but largely by the damping action of fluids in the tympanum.

f. Discussion and Conclusions on Acuity. The commonest activity in which pressure changes arise is high altitude flight; and much of the literature on aerotitis media has been written by flight surgeons.[1, 4, 8, 12, 13, 19, 21] Auditory acuity during flight, however, is complicated by at least two factors, the possibility of an actual difference in pressure across the tympanic membrane, and auditory fatigue. It is difficult to design an experiment using actual flight conditions which provides an accurate index of loss of acuity as the result solely of pathological changes in the ear. These difficulties are of course well known to flight surgeons.

But even when disturbing factors are controlled, it appears that our incidence of deafness is considerably less than that reported from the Air Forces. The difference must be explained in terms of the different barometric conditions which prevail between the two services. Although the pressure differentials are much greater in the Submarine Service (for it is self-evident that no pressure greater than 1 atmosphere need ever be considered in the Air Force), yet it is the relative differential rather than the absolute value which is significant. Moreover, we have shown[17] that the large majority of men who cannot sustain submarine pressures ask to be excused at pressures no greater than those to which aviators are commonly subjected. The main difference between submarine and air force

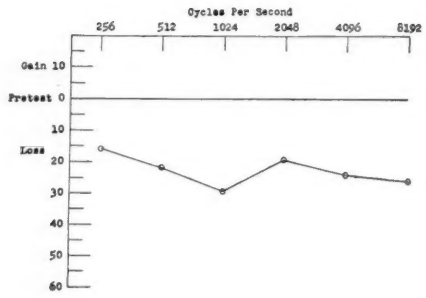


Fig. 11.—Difference between pre- and post-pressure audiograms for all No. 5 ears.

pressure conditions is that the usual flight consists of a decompression followed by return to normal pressure, while submarine training consists of a compression followed by a return to normal pressure. In the case of submariners, then, it will be seen that the negative pressure in the tympanum which produced vascular engorgement and rupture during the first or compression phase, changes to positive pressure during the second or decompression phase, and the result is that the vascular system tends to shrink and a form of therapy is achieved. The reverse is true for the aviators, in whom the second phase is one of compression, the men reaching the ground at a time when symptoms are most pronounced. One might for these reasons expect a greater incidence and severity of pathological changes and loss of hearing acuity among aviators than among submariners.

SUMMARY

1. The present study attempted to discover the causes and effects of aerotitis media and to find the best means of prediction, prevention, and treatment. In the course of these experiments 6,149 submariners were given 50 pounds air pressure. They were examined before and after pressure by means of the otoscope, nasopharyngoscope, and audiometer. Conclusions of this paper are based upon objective findings only.

2. A group of men who did not contract aerotitis media on a first pressure test was given a second test. Another group of men who did contract it was given a second pressure test with no treat-

ment intervening. These two groups served as controls for five experimental groups given different types of treatment as follows: psychological, topical, x-ray, radium, and dental. The types of treatment were all based on some rationale designed to assist the men in taking pressure in the future.

3. Psychological treatment included additional motivation, the use of chewing gum, and the use of music. None of these reduced the incidence of aerotitis media on subsequent pressure tests.

4. Topical treatment consisted of five drops of .25% neosynephrine in physiologic sodium chloride solution, applied to nose and nasopharynx every two hours for several hours before a second pressure test. Only a slight beneficial effect resulted.

5. X-ray therapy was discontinued and results were inconclusive.

6. Radium therapy consisted in the application for eight to ten minutes to the pharyngeal orifice of the eustachian tube of a small monel metal cylinder containing 50 mg. of radium salt. The cylinder, handled by a wire brazed to one end, was 2 cm. long, inside diameter 1.7 mm., with walls .3 mm. thick. The dose is effective, after two to eight applications each separated by a month, in reducing the amount of lymphoid tissue around the opening of the tube. Ninety per cent of the patients thus treated became able to sustain pressure without developing aerotitis media.

7. Dental therapy was investigated in 50 cases where improper jaw motion was suspected of hindering normal operation of the eustachian tube. Striking success in 46 of these patients was achieved by Kelly, who has reported his findings elsewhere.

8. Nothing was found to predict whether a given man would develop aerotitis media, except a history of repeated attacks. If a man were very poor in a preliminary Valvalsa maneuver, the chances were that he would develop the disorder. Positive relations were established with the appearance of the eustachian tube, whether open, flat, closed, or covered by lymphoid tissue, and also with size of adenoids; but the magnitude of the relationship did not permit of good prediction in individual cases.

9. Pain was not a good indicator of loss of auditory acuity.

10. Rupture of the tympanic membrane produced an acuity loss of 5-10 decibels.

11. Almost no effect on acuity was found unless the middle ear was filled with blood unmixed with air. Deafness as a result of

pressure is caused in submariners more by damping of the ossicles than by pathological changes.

12. Differences in pressure conditions and in effects of aero-otitis media between the Submarine Service and the Air Forces are discussed.

The broad details of this study were conceived largely by Captain C. W. Shilling, (MC), USN, to whom we are indebted for many suggestions and for providing all necessary facilities.

For much of the anatomy and for other helpful suggestions we are indebted to Dr. Stacy Guild, Johns Hopkins Medical School.

The suggestion for using music in psychological therapy was made by Comdr. H. Berman, (MC), USNR.

SOUND LABORATORY
MEDICAL RESEARCH DEPARTMENT
U. S. SUBMARINE BASE

REFERENCES

1. Armstrong, H. G., and Heim, J. W.: The Effect of Flight on the Middle Ear, J. A. M. A. 109:417-421, 1937.

2. Beaven, C. L.: Chronological History of Aviation Medicine, Flight Surgeons' Topics 2:185-206, 1938.

3. Boot, G. W.: Caisson Workers' Deafness, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 22:1121-1132, 1913.

4. Campbell, P. A., and Hargreaves, J.: Aviation Deafness, Acute and Chronic, Arch. Otolaryng. 32:417-428, 1940.

5. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. 112:585-590, 1939.

6. Crowe, S. J., and Burnam, C. F.: Recognition, Treatment and Prevention of Hearing Impairment in Children, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:1-17, 1941.

7. Farrior, J. B.: Histopathologic Considerations in Treatment of the Eustachian Tube, Arch. Otolaryng. 37:609-621, 1943.

8. Fowler, E. P., Jr.: Radon Treatment for Otitis due to Barotrauma, J. Laryng. and Otol. 58:489-492, 1943.

9. Haines, H. L.: Aero-Otitis Media in Submarine Personnel, J. Acous. Soc. Amer. 17:136-138, 1945.

10. Harris, J. D.: Auditory Acuity in Severe Aero-Otitis Media, J. Acous. Soc. Amer. 17:139-143, 1945.

11. Kelly, W. J.: The Results of Dental Therapy in Fifty Cases of Aerotitis Media in Submarine Personnel Based upon a New Functional Concept of Eustachian Tube Blockage. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 55:13-28, 1946.

12. McGibbon, J. E. G.: Aviation Pressure Deafness, J. Laryng. and Otol. 57:14-22, 1942.

13. Poppin, J. R.: The Ear in Flying, Laryngoscope, 51:974-982, 1941.

14. Rentschler, H. D., and Settle, J. W.: Treatment of Impaired Hearing by Radiation of Excessive Lymphoid Tissue in the Nasopharynx, Pennsylvania M. J., 47:985-988, 1944.

15. Requarth, W. H., Aero-Otitis Media in Compressed Air Workers, J. A. M. A. 116:1766-1769, 1941.

16. Rich, A. R.: Physiological Study of the Eustachian Tube and its Related Muscles, Bull. Johns Hopkins Hosp. 31:206, 1920.

17. Shilling, C. W.: Aero-Otitis Media and Loss of Auditory Acuity in Submarine Escape Training, Arch. Otolaryng. 42:169-173, 1945.

18. Shilling, C. W., and Everley, I. A.: Auditory Acuity in Submarine Personnel, U. S. Naval Medical Bull. 40:664-686, 1942.

19. Simpson, J. F.: A General Survey of Otorhinological Considerations in Service Aviation, Jour. Laryng. and Otol. 57:1-7, 1942.

20. Teed, R. W.: Factors Producing Obstruction of the Auditory Tube in Submarine Personnel, U. S. Naval Medical Bull. 42:293-306, 1944.

21. Yearbook of Eye, Ear, Nose, and Throat, 1941, S. J. Crowe, Ed., Chapter by Campbell, P. A., "Aviation Medicine," Chicago, Yearbook Publishers, 1941.

XXXII

THE PROBLEM OF SECONDARY FRONTAL SINUS SURGERY

FRANCIS L. WEILLE, M.D.

BOSTON, MASS.

At the Massachusets Eye and Ear Infirmary during 1944 a patient who had had eight external frontal sinus operations (four on each side without obliteration) came under the care of the writer. He was being given sulfadiazine for recurrent swelling, redness and tenderness of the right orbit. This occurred several days of each week in spite of a right Lynch frontal revision which had been meticulously carried out by one of our staff members a few weeks previously. Closure of the nasofrontal opening with scar tissue had occurred and the ethmoid area was occupied by scar tissue which involved the septum. A further revision was done and the patient recovered without obliteration of the frontal sinus. He has been followed for a year and a half and has had no further difficulty, apparently because he has a patent nasofrontal opening measuring about 3 by 8 mm. He has gained 30 pounds or more in weight and has survived without incident several head colds and has had no further chemotherapy other than locally.

The presentation of this case at staff meetings aroused a good deal of argument about the desirability of proceeding at once with obliteration of the affected frontal sinus (or of both frontals, since a small sequestrum had been found by x-ray study on the other side; this disappeared in later films). Lively discussion also arose regarding the use of the bell-end of a large, soft rubber French catheter which was left in the nasofrontal opening for seven and a half weeks, since many reasonable criticisms can be levelled at this method.

Four other "secondary" external frontal surgical cases seen at about this same time caused further debate as to methods. (For example, it was stated by one surgeon that obliteration never failed if

---

From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary.

Presented before the American Laryngological Association, Chicago, Ill., May 29-30, 1946.

there were no osteomyelitis.) It seemed worthwhile, therefore, to look up just what had happened with other similar patients in the previous fifteen years' experience (1930 to 1944) at the Infirmary, since this would offer facts and figures instead of more or less isolated impressions regarding the problem of secondary frontal sinus surgery.

Incidentally this period divided itself almost equally into years when chemotherapy was unknown, and years when it developed to approximately its present state.

During these 15 years 294 patients underwent external frontal sinus surgery. By omitting those with carcinoma and lost or misfiled records 276 were included in the survey. These patients had had a total of 862* operations upon the nasal cavities, sinuses and cranium, including plastic and brain procedures. Of these, 447* were directly upon the frontal sinus or sinuses externally, including 47 which were bilateral and 8 cases in which one frontal was done at one time and the other later. Forty-two patients died; a mortality of 15%.

Ninety-five of the patients had more than one external operation on one or both frontal sinuses. The highest number on one patient was nine, five on the right and four on the left, in the case described in the beginning of this paper. One hundred eighty-one of the group did not have either frontal sinus operated on more than once during the time they were followed at the Infirmary. These patients included those having bilateral procedures done at one operation, and those having one side done at one time and the other later. This group does not take into account any other procedure other than direct attack on the frontal sinus. Thus it deletes incision and drainage of the orbit before or after frontal surgery, secondary resection of osteomyelitic bone of the cranium, exploration for or drainage of a brain abscess or a subdural abscess.

The average total of operations per patient was 3.1, while the average of frontal sinus operations per patient equalled 1.6. The average length of time of follow-up was 67.2 weeks, measured from the time of the last (or only) frontal operation until the patient was last seen. In some cases the follow-up period was very short, and in others many years.

Since obliteration of the frontal sinus is so relied upon by surgeons of wide experience in secondary frontal sinus surgery, it

*Many of these operations were done outside the Infirmary, the patient being referred there as a "last resort".

might be well to discuss plastic procedures in this study as a preliminary to considering obliteration. The rhinologist usually employs a skilled plastic surgeon postoperatively to restore the patient's appearance and alleviate his mental reaction to deformity. By a few facts and figures one can properly evaluate the disadvantages of the disfigurement which causes hesitance in the adoption of the method.

Seventy-one cases of the 276 required plastic repair. One hundred eleven plastic operations were done on these patients. The average time from the last frontal operation to the last plastic procedure was 48 weeks. This average was weighted by cases in which simple closure of an open wound was done a few days after a frontal operation to those requiring months or years of plastic repair. In one instance, for example, five years three and a half months were required for three plastic operations. Three other cases required over four years for such work, while 11 cases required one and a fraction to two and a fraction years. These figures do not take into consideration the adverse mental reactions which may have occurred in some of the cases.

The purpose of obliteration is to seal off the frontal sinus communication with the nasal cavity and to obliterate any air space in the sinus itself. Obliteration is supposed to be a solution for frontal sinuses which have failed to do well after repeated efforts to establish drainage into the nose. The operation is designed to collapse the soft tissues of the anterior wall of the sinus against the bare bone of the posterior wall or the dura; that is, the anterior bony wall is always excised completely in any form of obliteration. Since the Lynch procedure removes the floor completely, this is often not a problem. Removal of the posterior bony wall offers theoretically a greater chance for successful obliteration since adhesions will form more readily, thereby, between the anterior and the posterior walls. The interfrontal septum must remain in unilateral obliterations and provides a mechanical source for failure, especially as the region of the frontal ostium lies very near the interfrontal septum posteriorly and adhesions must seal off all communication between the frontal sinus and the nasal cavity. If left behind, any bony wall of the frontal sinus, including the interfrontal septum, is also a source of possible regeneration of mucous membrane. If the ostium seals off and the mucous membrane regenerates, a mucocele is likely to develop.

The obliteration of both frontal sinuses simultaneously is supposed to offer advantages for success since the interfrontal septum is out of the way. Obliteration has the virtue of utilizing the tend-

ency of some frontal sinuses to shut off entirely any drainage into the nose after the operation because of scar tissue formation.

A wide variety of obliterative operations have been tried including those mentioned above and initial obliterations have been done without any previous frontal sinus operations where only a little of the floor is excised.

Results in 96 Cases of Osteomyelitis and 4 Cases of Osteoporosis. In the total group of 276 patients there were 96 who had osteomyelitis. In 44 the disease process extended beyond the limits of the frontal sinus into the cranium while in the remainder the disease was limited to the frontal sinus itself. Fifty-three of the group had a single operation directly upon the frontal sinus or sinuses, while 43 had multiple operations. Four of the 43 had multiple frontal sinus operations without obliteration; 14 had frontal sinus surgery followed by obliteration; 25 had obliteration followed by more surgery and 8 of the 25 had external frontal sinus surgery before obliteration.

A total of 77 patients had obliterative operations; 15 of the 77 had re-obliteration; 7 more had other frontal sinus surgery after obliteration, while 3 others had incision and drainage of the orbit (once in each case) after obliteration.

Of the 15 patients who had re-obliteration the original obliteration procedure in every case was removal of the anterior wall and the floor. All of the 15 had the posterior wall removed at re-obliteration and in 13 of the 15 cases obliteration was the first procedure.

Of the 7 patients having further frontal sinus surgery after obliteration: 3 had initial removal of all walls bilaterally; one had bilateral removal of the anterior and posterior walls and the floor of one side. All 4 of these cases were re-operated, 3 once each and the fourth twice more. The remaining 3 (of the 7 patients) had unilateral obliteration with the anterior wall and the floor removed. One of the 3 had five further frontal sinus operations on the same side plus incision and drainage of the orbit and drainage of a brain abscess. The remaining 2 patients had one further operation each.

Three patients had simple incision and drainage of the orbit (once each) after obliteration. In 2 of these, the anterior wall and the floor had been removed, bilaterally in one and unilaterally in the other. In the third the anterior and posterior walls had been removed on one side. In summary, 25 of 77 cases of osteomyelitis required

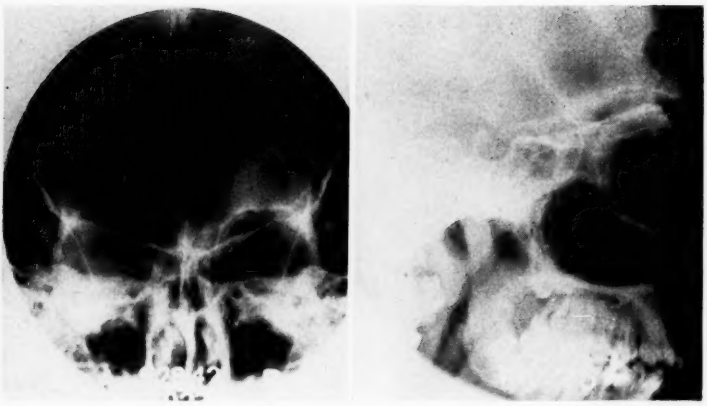


Fig. 1.—Roentgenograms showing excision of frontal sinuses and frontal bone for osteomyelitis.

direct frontal sinus surgical procedures or incision and drainage of the orbit after obliteration operations.

Tables 1, 2, 3, and 4 summarize the above facts, including the types of surgical procedure and the mortality, while Table 5 epitomizes the group of osteoporosis cases.

The purpose of reviewing carefully the experience with osteomyelitis is that the seriousness of the disease process and the high mortality may justify surgical decisions, especially for obliteration, which might not be tenable otherwise. Since approximately one-third of the obliterative cases required further surgery in osteomyelitis, it becomes of interest to study the facts and figures obtained in the non-osteomyelitic group.

Results in 176 Non-Osteomyelitic Cases. Of the remaining 176 cases, only 52 patients had multiple operations. Table 6 summarizes some of the more interesting findings, including the fact that of 23 who had obliteration, 8 required further surgery. Table 7 gives further details regarding these 23 patients, while Table 8 emphasizes the remarkable finding that only 29 patients of 276 had no osteomyelitis, no obliteration, and repeated operations on the frontal sinus itself. That is, of 176 who had no osteomyelitis, 52 had multiple operations, divided 23 with obliteration and 29 without.

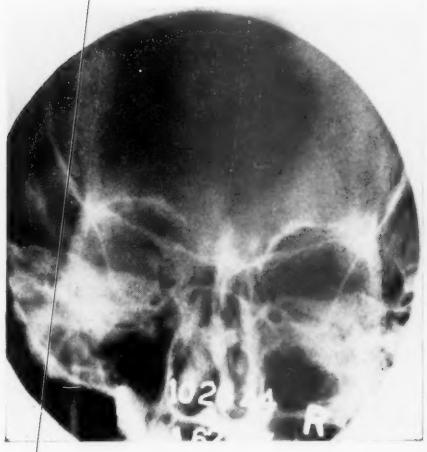


Fig. 2.—Roentgenogram showing postoperative bone regeneration.

The type of surgery and the mortality occurring in the 124 patients with one operation on one or both frontal sinuses is summarized in Table 9. It is of interest here that 17 of 23 patients having obliteration by removal of the anterior wall and the floor had no further operations during the Infirmary follow-up period, and that the 5 who had excision of the floor, the anterior and the posterior walls all died. (This was not related to simple removal of the posterior wall, but to the fact that these were complicated cases.)

While the Lynch operation was the commonest in the survey (191 Lynch operations or Lynch-type "revisions" were done), a total of 123 obliterative cases places the obliteration operation second in frequency.

Causes of death in the total group were meningitis, subdural abscess, brain abscess, thrombophlebitis and septicemia, pneumonia and in one case pinealoma.

COMMENT

It seems obvious from the facts and figures stated that the acceptance of obliteration as a choice of surgical procedure is not an unmixed blessing even when plastic repair and the mental state of the patient as a reaction to deformity are completely discounted.

On the other hand, the alternative fact that 29 patients requiring "secondary" frontal sinus surgery but not having obliteration underwent 77 operations is discouraging. Is the surgeon confronted with the dilemma of choosing between obliteration and its drawbacks, or the probability of having to "revise" a secondary frontal operation repeatedly? The causes for failure of an ordinary Lynch frontal operation have been listed by Goodale[1] as: adhesions; remnants of the floor of the frontal sinus; residual ethmoidal cells, particularly a fronto-ethmoidal cell or an ethmoid extension into the roof of the orbit.

To these may be added, closure by growth of bone. The writer recently found at a secondary frontal operation a mass of bone 2 cm. in diameter causing complete closure between the medial portion of the frontal sinus and the anterior ethmoid, with total obliteration of the former surgical opening.

The "remnants" of the frontal floor found at a secondary operation are not infrequently simple regeneration of bone, and it is pertinent to challenge the validity of the hallowed axiom among rhinologists that a completely excised frontal sinus floor never regenerates.

In Fig. 1 is seen a postoperative x-ray film of a 14-year-old girl who had osteomyelitis of the frontal bone excised along with all walls of both frontal sinuses through a coronal incision with immediate closure of the wound. She had previously had a left Lynch frontal. The only drainage after the frontal bone excision was through this wound. She was discharged on the thirty-second day with the wounds completely healed.

Figure 2 shows a further x-ray film taken two years later. The radiologist reported the lower third of the bony defect to have regenerated and the frontal sinuses to have reformed.

The most important reason for failure in non-obliterative external frontal sinus surgery—primary or secondary—is simple closure of the nasofrontal opening. Can this unhappy situation be improved? Several considerations are in order; they are: applied surgical anatomy, the process of wound repair in this region, methods of keeping the nasofrontal opening patent, avoidance or simplification of surgical treatment when possible, and chemotherapy. These will be discussed in this order.

Applied Surgical Anatomy. Since the normal ostium lies posteriorly and medially near the crista galli, certain anatomical facts should be kept in mind in attempting to establish a surgical opening

into the nose. The first of these is that the ethmoid is the normal surgical pathway into the nose from the frontal sinus; that it extends anteriorly only as far forward as the ascending process of the superior maxilla. That is, a large part of the nasal part of the frontal bone is in relation with the ascending process and the nasal bone laterally and anteriorly. There is merely a 3 mm. wide groove in the roof of the nasal cavity at the floor of the frontal sinus. The floor of the frontal sinus anteriorly and medially consists of heavy thick bone which tapers posteriorly to thin bone in the region of the frontal sinus ostium. If an attempt is made to establish a pathway between the floor of the frontal sinus anteriorly and the nasal cavity, anatomically the space is narrow and thick bone must be tunneled permanently.

In terms of healing, the establishment of the nasofrontal opening in the ethmoid relationship has many added advantages: The ethmoid is, according to Mosher[2], 5 mm. wide anteriorly and widens further to 15 mm. at the face of the sphenoid. The greater the depth of the frontal sinus and the greater its ethmoidal relationship, the greater is the possibility for utilizing ethmoidal space from before backward and from side to side for the nasal opening. The 3 mm. groove beside the septum at the roof of the nose on the floor of the frontal sinus adds just so much space to that gained by utilizing the ethmoidal anatomical relationship. Furthermore, a large frontoethmoidal cell in series with the posterior wall of the frontal sinus medially would seem to offer anatomically definite aid in making a Lynch operation successful by utilizing it as an anatomical pathway for drainage after its anterior wall had been excised completely. In the fundamental healing process, it should be remembered that the "large opening" which the surgeon makes from the frontal sinus into the nose in the Lynch operation becomes anatomically very much smaller when the wound is closed.

The Process of Wound Repair in Secondary Frontal Sinus Surgery. The tissues concerned in postoperative repair are:

1. Bone denuded of mucoperiosteum. The denuded bony walls of the frontal sinus and the ethmoid cells have, even with "complete" removal of the sinus lining, microscopic fragments of mucoperiosteum from which regeneration of the sinus lining takes place.[3-5]

2. True bare bone. Most important is a wedge of freshly cut raw bone lying between the frontal septum superiorly and the nasal septum inferiorly. This is in relation, in a skillfully performed operation, inferiorly with the intact mucoperiosteum of the septum.

It has no diploe.

Adult bone cells here as elsewhere will not proliferate; bone repair therefore proceeds from osteoblasts of the haversian canals, as well as from "neighborhood" osteoblasts including those of the nasal mucoperiosteum and islands left on the frontal sinus septum and other walls of this sinus.

3. Mucoperiosteum of the nasal cavity.

4. The diploe, if the operative field involves the anterior wall of the frontal sinus and the uppermost portion of the posterior wall, or the "outer angle" of the frontal. (In an ordinary Lynch frontal operation, the diploe is avoided, or almost avoided).

5. The orbital periosteum, and that of the lateral external nose.

6. The skin, subcutaneous tissue, and orbicularis oculi muscle.

The most important "common denominators" for repair are, therefore:

1. Epithelium.
2. Connective tissue stroma and its contents.
3. Bone.

A few facts regarding these tissues can be briefly but profitably reviewed.

Kazanjian teaches that soft tissue contracture occurs near bone in a certain orderly manner as follows: If granulation tissue is completely encircled by bone, it tends to contract toward the bony circumference and away from the center of the circle. Granulation tissue which is surrounded solely by soft tissue tends to contract centrally, pulling in the surrounding parts toward the center of an imaginary circle. There are wide degrees of variation between these two extremes but the greater the amount of a circle of bone which can be maintained around an intended lumen, the less likely is the opening to close. The practical application of this point is that the surgical communication between the large frontal sinus cavity and the large naso-ethmoidal cavity has to be made at a relatively narrow point and that the wide removal of bone in the region of the upper portion of the ascending process and the nasal bone or the contiguous portion of the nasal part of the frontal bone lessens the periphery of the partial residual circle. This sacrifices a valuable adjunct in healing in direct proportion to the amount of bone lost. In other words, the junction of the posterior inferior margin of the frontal sinus with the orbital plate of the frontal bone

constitutes part of the bony circle; the wedge of bone lying between the nasal septum and the frontal septum, another part of the circle; while the bone just described constitutes what is left of the circle anteriorly. Inasmuch as the technique of the Lynch frontal operation requires the destruction of the lateral portion of the circle, the area of such destruction should be as limited as possible so far as is compatible with adequate procedure. Furthermore when bone is widely sacrificed, the disadvantage should be known to the operator before he does it, rather than afterward. This does not mean retention of any portion of the floor of the frontal, or failure to have a smooth surface of bare bone between the frontal septum superiorly and the nasal septum inferiorly.

The last is quite important, as bare smooth bone is ideal for the growth of epithelium from the well-preserved nasal septal mucoperiosteum upward onto the frontal septum. The growth tendency of underlying granulation tissue (whether osseous or connective tissue granulations) is restrained by a covering of epithelium. Furthermore, everyone recognizes that adhesions will not form between intact sheets of epithelium and overlying granulations.

The sources for epithelial regrowth are: the undisturbed mucosa of the nasal cavity and microscopic islands left in the ethmoid and frontal when the bone is denuded. It is known that in the denuded antrum epithelization is unmistakably present only at four weeks.[5] It is therefore imperative for immediate postoperative repair that the undisturbed nasal mucous membrane not be traumatized beyond the field required for adequate surgery.

Arey[6] believes the restoration of epithelial defects "is brought about by the movement of epithelial tissue from the intact border zone over connective tissue or other substrate. Advancing from all sides these modified reparative epithelial sheets meet and then return to their normal state of organization." Conditions which alter this process and its time relationships are "the size and shape of the wound; the preparation of a proper bed in secondary repair; the occurrence of mechanical obstacles; the presence of infection; the kind of epithelium involved (simple or stratified)."

The extension of epithelium is in progress even on the first day. In the first two weeks the epithelial tongues adhere more firmly to a wound scab than to underlying connective tissue, so that lifting up of the scab tends to carry the epithelium with it.

Hajek is said to have devised a surgical technique for observing directly the process of epithelial repair of the nasofrontal passage-

way following radical external frontal operations. Hajek taught his pupils that three to six months were required for complete healing.

Since epithelial repair in mucous membrane proceeds in approximately the same manner fundamentally as in skin,[6] it is interesting to know that skin epithelial growth is at the rate of 0.5 mm. per day[7] and begins after a latent period of three to six days. Frequent dressings prolong the latent period by tearing away epithelial cells as they advance. In early weeks the rate of repair is constant; that is, the growth of skin epithelium is not rapid for a few days followed by slowing down. Local conditions in the wound influence the amount of daily extension of epithelium; for example, infection stops epithelization entirely.

Connective tissue repair is described by Arey.[6] The cells involved are neutrophiles, polyblasts, fibroblasts, and endothelium. Polyblasts, which are said to arise from lymphocytes, monocytes, and histiocytes may revert to histiocytes (reticulo-endothial macrophages) or be transformed into fibroblasts. In relatively clean wounds, fibroblasts and endothelium are proliferating plentifully—the latter as vascular sprouts—on the second day. While the great source of fibroblasts is proliferation by fibroblasts, these cells may come, in later healing, partly from endothelium.

Infection, necroses, foreign bodies, and blood clots are adverse factors in the growth of healthy granulation tissues. Blood stasis tends to produce thick weals of tissue.

A "granulation" has a vascular tree as a core with an enveloping mantle of young fibroblasts and wandering cells.

Pressure sufficient to blanch a fingernail, when applied to granulation tissue, tends to lessen its growth by diminishing vascularity. Such pressure still allows epithelial growth to take place.

The laying down of collagen fibrils is largely responsible for wound contracture in the *late* stages. It is this contracture which results in the closure of an apparently good nasofrontal opening several months after operation when success has seemed assured. The use of any form of obturator in a soft tissue stoma in which no epithelial covering occurs usually fails to prevent closure by contracture. Possibly elastic fibrils which appear from about three to nine months after the onset of soft tissue wound healing play a part in late contracture, but the collagen fibrils with which everyone is familiar are all-important. Aging collagen contracts[8] through losing water; presumably the water is as closely related to the protein collagen as water of crystallization is to a crystal.

It is known[6] that contracture occurs *early* in wounds of skin and mucous membranes which are loosely attached to deeper structures. Carrell is cited[6] as finding that contraction begins after a quiescent period of two to five days in large and medium-sized wounds and proceeds fastest immediately thereafter, followed by gradual slowing to a standstill. Some mechanical factor such as shrinkage is presumably the cause. The more firmly anchored the soft tissue is to deeper rigid structures, the less important is this early contracture, and it does not occur in wounds less than 10 mm. in size.

Bone healing is less clearly understood than is soft tissue repair.

Normal bone metabolism has been briefly outlined by Reifenstein and Albright[9] as follows: Normal bone consists of an organic matrix in which a calcium-phosphate-carbonate salt is deposited. On normal bone there are areas where nothing is happening, where bone is being formed, and where bone is being resorbed. In the last area osteoclasts are seen. Bone formation has two steps: The laying down of the matrix by osteoblasts; and the deposition of calcium salt. Bone formation and bone resorption occur simultaneously. This is probably made possible by the enzyme phosphatase, which is said to be produced by osteoblasts. This enzyme increases the local concentration of phosphate ions by splitting off such ions from organic phosphate compounds, and thereby permits the deposition of calcium, phosphate, carbonate salt. Calcium metabolism is regulated by the parathyroid glands and vitamin D.

No tissue in the body is capable of so much overgrowth on one hand or absorption on the other as bone.[10] Proliferation of osteoblasts may be so rapid and massive that no other nonmalignant process is comparable with it.

The raw bone surfaces in a postoperative frontal sinus are bathed by blood and exudate. Osteoblasts from the mucoperiosteum and from the haversian systems would in normal bone repair join with new capillaries in forming a kind of granulation tissue. In four or five days trabeculae would be formed around central spaces—later becoming haversian canals. This is osteoid tissue which with calcium deposition becomes bone.

But in the Lynch frontal operation, the purpose is to defeat such normal healing and it is postulated that normally, after complete removal of the floor of the frontal sinus, no such bone regeneration occurs. This postulate is of doubtful merit, as has been pointed out above.

In the Lynch frontal operation when the wound is closed, periosteum is sewed to periosteum. What is to prevent osteoblastic activity? It is true that bone regeneration is irregular, especially in membranous bone. To secure regeneration: Get a great big blood clot. Strip the cambium layer of bone with the periosteum, making the latter look pebbly, and using extremely sharp periosteal elevators. Then when the periosteum is sutured to periosteum, bone may regenerate, as osteoblasts cause secretion of osseomucin and phosphatase. Prevention of regeneration may possibly be avoided in proportion to the accomplishment of the opposite of these measures.

In summary, fundamental tissue healing in relation to epithelium, connective tissue and bone has been briefly reviewed with the particular purpose of seeing what aid could thus be obtained in establishing a permanent nasofrontal opening. While many factors may make for failures requiring secondary frontal surgery, the most important one is simple closure of the stoma between the nasal cavity and the frontal sinus. If the frontal sinus is regarded as one circle in a figure of eight and the nasal cavity as another circle, it is vital to have a patent opening between the two at the anatomical hourglass constriction.

Methods of Keeping the Nasofrontal Opening Patent. 1. Anatomically there is reasonable likelihood that many Lynch frontal operations will succeed, particularly if the patient is fortunate enough to have a large medially placed fronto-ethmoidal cell which, when made a part of the frontal sinus by complete removal of their common wall, allows drainage near the region normally occupied by the frontal ostium. Such an opening may occur without the use of any foreign body as a drain or obturator. Some surgeons believe that the irritation resulting from the use of a foreign body such as a catheter induces a damaging granulation tissue response.

2. However, it was Lynch,[11] himself, who left in a soft rubber drainage tube three-eighths of an inch in diameter with one end cut on a long bevel, taking the tube out at the end of five days. He then used a large dilator for ten days more and concluded that then "healing seems to be established and convalescence about complete." Schall modified this technique by using the bell end of a large soft rubber French catheter. He placed the bell end in the frontal sinus and the small end was allowed to protrude from the nasal vestibule where is was secured externally. Such a catheter has the advantage of acting as a drain and of permitting irrigation of the frontal sinus with penicillin solution or other chemotherapeutic agents. It produces a minimal granulation tissue response if the bell end fits well into

the nasofrontal opening. The fact that it is not a non-irritating foreign body can be minimized so far as healing responses are concerned by leaving it in for a very few days after Lynch's technique or permitting it to remain deliberately for many weeks so as to obtain its maximum value as an obturator. As has been stated above, failure of epithelial growth around the catheter will in the end permit enough contracture of connective tissue to cause failure.

The method worked well in the patient who had the highest number of Lynch revisions in this survey of 276 cases.

An attempt by the writer to use a paraffin-covered catheter was successful in establishing drainage into the nose in dealing with a postoperative mucocele in which 2 cm. of solid bone occluded what had once been a Lynch frontal opening into the nasal cavity. The tube was placed posteriorly into the ethmoid behind the occluding bone and it was hoped that the paraffin might mitigate some of the ill effects produced by soft rubber. Meltzer had suggested this technique as a result of experience with the fenestration operation and stated that Holmgren had successfully employed paraffin originally in preventing bone regeneration.

3. Mucous membrane flaps have been frowned upon by the majority of surgeons dealing with frontal sinus surgery. Sewall[12] reported the successful use of such flaps many years ago. The writer has occasionally employed a flap from the upper anterior lateral nasal wall by turning the flap onto the frontal septum like a hinge. A great disadvantage is the maintenance of the flap in situ. The method was dramatically successful in the following case:

A 48-year-old white male had had a right external frontal operation in London in 1928 which had been repeated there in 1935. From then until the time he was seen in 1942 he had had recurrent orbital abscesses with closure of the right eye for about a week out of each month. The frontal sinus was explored and "revised"; complete closure of the opening between the frontal sinus and the nasal cavity was found to have occurred. A mucous membrane flap was turned from the lateral nasal wall against the frontal septum at the operation. At the time of his discharge he had a widely patent opening from the nose into the frontal. The patient was last heard from in April 1945 when he reported that he was completely well and that he had had no trouble since the operation.

4. Another method is probing. Every experienced rhinologist has occasionally been chagrined to observe, over a course of months, what seemed like a perfectly successful secondary nasofrontal opening (or primary opening for that matter) slowly close in spite of every effort in local wound management. He is then compelled periodically to dilate the opening with probes to try to keep at least an intermittently patent fistula present. The process is uncomfor-

able and troublesome for the patient and in the hands of the writer has been of only palliative value.

5. Skin grafts have been found useful by some rhinologists. Smith has reported[13] their use in over 100 cases with only three closures of the nasofrontal opening.

6. A sixth method is the use of non-irritating foreign bodies. It should be pointed out that the distinction between irritating and non-irritating foreign bodies as used in the frontal region needs careful understanding. It is of prime importance that no dead space exist around a foreign body of either type; this insures the least tissue response in either case. If a French rubber catheter fits well in the nasofrontal opening, the slight pressure which it exerts upon surrounding granulation tissue lessens the blood supply of the granulations and thereby lessens their growth, while at the same time this pressure is insufficient to stop or prevent epithelization. If, for example, an acrylic resin tube were substituted for the catheter, it would excite a foreign body response if its fit were poor even though the tissues have great tolerance for methyl methacrylate.

Non-irritating foreign bodies which have proved useful in frontal sinus surgery include tantalum,[14] acrylic resin and vitallium.[15] To this list may be added fibrin film as a promising substance for trial.

In using foreign bodies to aid in establishing a permanent nasofrontal opening, the material must be sterilizable; it must cause as little tissue reaction as possible; it must check the growth of granulation tissue (whether osteogenic or fibrogenic) throughout the period required for healing of the operative field. Furthermore, the material must be easily available. If absorption of the foreign body occurs, the period must be relatively prolonged. Some of the properties of fibrin film, vitallium, acrylic resin, and tantalum will be outlined.

(a.) Fibrin Film. The material is prepared from human fibrinogen and thrombin and is dispensed as a thin firm translucent, rather brittle, dry, sterile sheet sealed in a glass ampule. After the film is soaked in cool, physiologic, sterile solution, it becomes flexible and elastic and should be used the same day the ampule is opened and should be kept immersed in cool, sterile, sodium chloride solution until it is required. It cannot be resterilized. If the surgeon believes there is danger of its slipping out of place it can be sutured as desired provided it is kept moist. It is completely absorbed within six months, but is still present in dural wounds at three months. Dr.

John Edsall of the Department of Physical Chemistry at the Harvard Medical School has recently made available to the writer sufficient fibrin film for a trial in frontal sinus work. A report about its use will be published later. The ability of this substance to prevent meningocerebral adhesions[16] would seem to offer hope for good results from it as a surgical aid in establishing a permanent nasofrontal opening.

(b.) Vitallium. Vitallium[17] is an alloy composed chiefly of cobalt and chromium, with a small percentage of molybdenum. Its specific gravity is 8.29. It is very strong and very light. It is boilable, and is said to be inert in body tissues.

Its use in strictures of the common bile duct[18] suggested that it might be of value in the surgical construction of a nasofrontal opening. Because the metal is not pliant or easily adaptable to molding in the operating room, an effort by the author to make use of a vitallium tube in frontal sinus surgery was abandoned.

(c.) Acrylic Resin. Harmon[19] describes the methacrylate resins as plastic materials having the following properties: They are polymers of methyl methacrylate. When cast in rods, sheets, or blocks the material is crystal clear, light in weight, with a specific gravity of 1.16. They are slightly flexible, and are durable and strong. Solid methacrylate is not affected by alkalies, oils, dilute acids, solutions of mineral salts, or dilute alcohol. Cast methacrylate softens between 190° and 240° F.; moldings withstand 140° to 190° F., depending upon whether prepared from "soft" or "hard" powder.

Irritating solvents called "plasticizers" used in industry are present only in traces in the methacrylates employed in surgery.

The tissues show great tolerance to the acrylic plastics. The material can be made into tubes of any desired size or shape and by using "soft" powder it is possible to mold the material as desired at the time of operation. It can also be made into a film upon a catheter as a coating in an effort to convert soft rubber into a nonirritating foreign body.

(d.) Tantalum. Tantalum is an element having an atomic weight of 180.88, a valence of 5, a specific gravity of 16.6, and a melting point of 2996° C. It is biologically inert and has the advantage of being an element, not an alloy as is vitallium. Chemically it is similar to glass in its resistance to acids. It is resistant to weak but not strong alkalies. Its chemistry is such that theoretically it should be inert

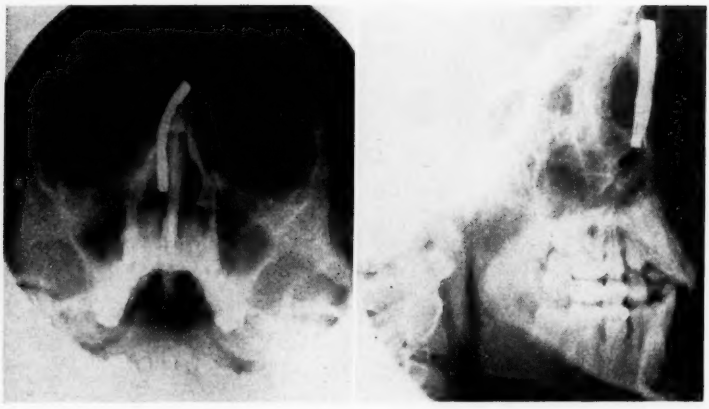


Fig. 3.—Roentgenograms showing tantalum tube in the frontal sinus.

in a body-tissue environment. This hypothesis has been borne out experimentally.

The metal is both malleable and ductile, so that it may be rolled into sheets of any desired thickness or drawn into extremely fine wire. The high tensile strength of the latter permits twisting and tying in tight knots. Very thin rolled sheets are called tantalum foil, which measures 0.00025 of an inch in thickness. Tantalum sutures are two and a half times stronger than the U.S.P. requirements for catgut (knot pull).

Tantalum combines the advantage of resistance to corrosion and chemical change with the advantages of the strength, workability, and malleability of steel.

Tantalum has been employed by the writer as a tube permanently implanted with one end in the frontal sinus and the other in the nasal cavity, using tantalum sutures and molding the tube to fix it in place.

Brief details of this case are as follows:

When the patient, a 9-year-old white male was first seen by the writer, his history was as follows: He had had left-sided, acute pansinusitis with orbital cellulitis. Decalcification of the outline of the left frontal sinus developed in spite of chemotherapy. A left external fronto-ethmoidectomy was done with complete removal of the anterior wall and floor of the left frontal sinus. The patient was well for six months, but then complained of severe pain in the left orbit,

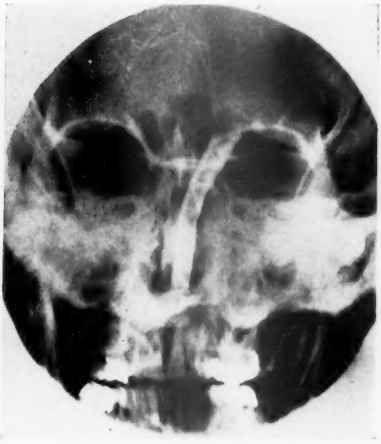


Fig. 4.—Roentgenogram showing tantalum foil covering a soft rubber catheter in the frontal sinus. A tantalum wire retention suture is seen also.

which was accompanied by swelling and tenderness of the forehead. The edema improved, but did not disappear with chemotherapy. X-ray films showed that the back wall of the frontal sinus up high was gone in two areas. Re-obliteration was carried out with excision of the posterior wall. Because of a small perforation in the interfrontal septum, this structure was removed entirely. His convalescence was uneventful, and he remained well for about eight months, except for recurrent swelling of the forehead with head colds.

Then, following swimming, he developed swelling, redness and tenderness over both frontal regions. An abscess of the left frontal area was incised and drained. About seven weeks later, a tantalum tube was placed by the writer in the right frontal sinus at its posterior wall just lateral to the margin formerly occupied by the interfrontal septum; the tube was brought into the left nasal cavity, and was fixed by tantalum sutures. Its position is shown in Fig. 3. Convalescence was uneventful. The patient has been well during a follow-up of one and a half years, despite several head colds.

Tantalum foil can be used to cover an ordinary soft rubber French catheter to make it relatively innocuous to tissues. Tantalum wire ties hold the foil in place. This was found useful in a case of tuberculous rhinitis complicated by right ethmoiditis and frontal sinusitis with orbital abscess, operated upon by the Lynch technique. It was possible for the patient to tolerate the tube, without difficulty, for four and a half months, when it was finally withdrawn. A tantalum retention suture through the upper end of the tube anchored it during this time. The suture, emerging through the wound, was

fixed to the forehead with Scotch tape and was completely innocuous also. The patient has required no further surgery to date, but while the tuberculous process has been slowly becoming dormant, meticulous care of the nasal cavity has been required. Fig. 4 shows x-ray of the tube in situ.

The Goodale operation[14] in which tantalum foil is sutured to the orbital periosteum has, in difficult cases, much to recommend it. It has the great advantage of relative simplicity. He has found no disadvantage from leaving tantalum foil permanently in situ in the eight cases reported by him.

Avoidance or Simplification of Surgical Treatment When Possible. Experience with radical external frontal sinus surgery has developed among rhinologists a wholesome respect for the frontal sinus and its complications. They require convincing evidence of necessity before surgical interference is undertaken. If the first external frontal operation can be avoided entirely by reasonable conservatism, the judgment and skill of the rhinologist deserve praise.

Moreover, Brown,[20] Walsh,[21] and others have advocated greatly simplified initial surgical techniques for dealing with many of the problems encountered. Thus, Brown has described simple trephination of the frontal floor with the establishment of external drainage in many of the acute cases requiring surgical intervention. He routinely orders adequate penicillin therapy throughout the infection.

In certain chronic frontal sinus cases Brown has trephined the frontal floor and renewed the partitions and the mucosa except in the region of the ostium. The wound is closed except for a drainage tube which is withdrawn when discharge from it ceases.

Simple trephination was undertaken initially in 7 of the 276 cases summarized in the first part of this report. In three of the patients there was uneventful convalescence; one had no chemotherapy; while the other two were given penicillin. Four patients required one to several further operations. Two of these had sulfonamide therapy, while the other two had none. There was one death—chemotherapy was not in use at the time of this patient's illness.

The preservation of the frontal ostium and the mucous membrane near it in the MacKenzie Brown operation for chronic frontal sinusitis is predicated upon clearing the nose of anatomical and pathological obstructions, together with preliminary studies ruling out allergy and endocrine dysfunction, and the failure of simple conservative treatment.

Since the nasofrontal communication is ordinarily not a duct[2] but an ostium in the floor of the frontal sinus postero-medially leading into an air space between the agger nasi and the lacrimal anterior ethmoidal cells, it is occasionally possible in intranasal ethmoidectomy not only to identify the frontal ostium but to preserve the mucosa on the ethmoidal side. This procedure alone will sometimes permit chronic frontal sinusitis to clear up.

It is highly desirable in "sinus" cases—especially in those in which external frontal surgery has been done or is contemplated—to make the nasal cavities as mechanically and physiologically perfect as possible relatively early rather than late in the course of the disease process.

The pathological, accidental or deliberate surgical perforation of the interfrontal septum is not always so serious as is commonly supposed. There is a sort of axiom among some rhinologists that such a perforation almost invariably means that the normal side if perforated will come to surgery. In only 5 of 18 cases in the present series in which such perforations occurred was a further operation done.

Chemotherapy. Chemotherapy, particularly penicillin therapy, has raised the hope that a great deal of frontal surgery can be avoided entirely or markedly simplified. While its aid is invaluable in grampositive infections at the present time, it is not a substitute for fundamental surgical principles.

Simpler surgical methods have proven satisfactory when combined with adequate chemotherapy. For example, at the clinical staff meeting of the Massachusetts Eye and Ear Infirmary on April 3, 1946, Fred reported a 25-year-old male patient who had acute left frontal sinusitis with superior sagittal sinus thrombophlebitis. The man was unconscious and having convulsions. The neck was stiff and there were 2200 cells in the sterile spinal fluid chiefly lymphocytes. Sulfadiazine was given together with penicillin intravenously, and 32,000 units of penicillin were given intrathecally three times a day. No operation was done for six days during which time the patient first became worse and seemed moribund, then recovered completely symptomatically. Electro-encephlagrams, which showed widespread disturbances over the frontal cortex of both sides, had become normal at the end of six days. Fred then carried out incision and drainage of the floor of the left frontal sinus, disturbing the mucous membrane only at the point of the entry into the sinus. A staphylococcus aureus was found in the pus contained in the sinus. He placed a tube

to but not through the opening in the frontal floor and irrigated the sinus systematically every few hours with penicillin. When he opened the sinus he was able to observe the point of probable entry of the infection into the meninges—posteromedially near the superior sagittal sinus. The tube was withdrawn after a few days, the wound closed with uneventful further recovery of the patient who had been followed about three months when the report was made.

Monto and Lyle[22] have reported a case of acute pansinusitis with orbital cellulitis, septicemia and meningeal irritation, with recovery by means of penicillin therapy, nasal shrinkage, and an orbital stab wound. The organism was a staphylococcus aureus.

Allman,[23] in a wide experience with scarlet fever, treated 52 cases of orbital cellulitis with penicillin given intramuscularly combined with the usual local therapy. None of these patients were operated on despite the fact that 12 had considerable exophthalmos, and 3 of these had complete loss of motion of the eye.

After the advent of chemotherapy, sulfonamide drugs and penicillin were used in the present series of 276 cases, in 82 of the patients. Prontylin, sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine were utilized in this sequence and then penicillin with or without the sulfonamides (sulfadiazine and sulfamerazine). It is of particular interest that only one patient died after sulfadiazine and penicillin were used.

SUMMARY AND CONCLUSIONS FROM A REVIEW OF 276 FRONTAL SINUS SURGICAL CASES

1. These patients had a total of 862 operations upon the nasal cavities, sinuses and cranium, including plastic and brain procedures. Many of these operations were done outside of the Massachusetts Eye and Ear Infirmary.

2. Of these, 447 were directly upon the fontal sinus or sinuses externally. Some of the patients were referred to the Infirmary as a "last resort."

3. 95 of the patients had more than one external operation on one or both frontal sinuses. The highest on one patient equalled nine.

4. Aside from deformity the obliterative operation is not an unmixed blessing. Thirty-three of 123 patients treated by this method required further frontal surgery.

5. The repair of the deformity required an average of about one year in 71 patients. The longest time was five years, three and a half months for such repair.

6. Only 29 of the 276 patients had no osteomyelitis and multiple non-obliterative operations (totalling 77) on the frontal sinus or sinuses.

7. A basic knowledge of applied surgical anatomy, and a fundamental understanding of epithelial, connective tissue, and bone responses in wound healing in the frontal region are helpful in voiding repeated frontal operations.

8. Simple closure of the nasofrontal surgical opening is a paramount cause for the necessity of secondary frontal sinus surgery. Some surgical methods for dealing with this problem are outlined.

9. The avoidance or simplification of surgical methods in the treatment of frontal sinus disease may be greatly aided by chemotherapy, especially penicillin. However, chemotherapy is not a substitute for surgical fundamentals.

10. Only one death occurred in the series after sulfadiazine and penicillin were used.

Acknowledgment is made to Dr. G. B. Fred for permission to quote his case.

247 COMMONWEALTH AVENUE.

TABLE 1.—OSTEOMELITIS.

	CASES	MORTALITY CASES
Total patients	96	23
Single operations	53	12
Mutiple operations	43	11
Total operations directly on frontal sinus in these cases—124. Average—2.9.		
Obliterative operations	77	20
Further surgery required after obliteration	25	6
(Total number of operations—31)		

TABLE 2.—FURTHUR SURGERY AFTER OBLITERATION IN OSTEOMYELITIS: 25 CASES.

	CASES
Type of operation at original obliteration:	
Removal of anterior wall and floor (bilateral in 7)	20
Removal of all walls bilaterally	3
Removal of anterior and posterior walls on both sides and floor on one side	1
Removal of anterior and posterior walls	1
Surgery at further operations:	
Removal of posterior wall	15
Incision and drainage of orbit	3
Miscellaneous	7
Mortality	6 cases.

TABLE 3.—SINGLE OBLITERATIVE OPERATIONS IN OSTEOMYELITIS.

Total patients 52

	ONE SIDE	BOTH SIDES	TOTAL
Type of Surgery Done:			
Anterior wall and floor removed	11	1	12
Anterior wall, posterior wall and floor removed	10	19	29
Anterior wall and posterior wall removed	4	5	9
Anterior wall, posterior wall and part of floor removed	0	2	2

Mortality ..... 5 cases.

TABLE 4.—OSTEOMYELITIS.

	CASES	MORTALITY CASES
Patients *not* having obliterative operations	19	3
Type of surgery:		
Modified Lynch	15	1
Miscellaneous	3	1
No surgery	1	1

TABLE 5.—OSTEOPOROSIS.

	CASES	MORTALITY CASES
Total patients	4	0
Type of operation:		
Lynch	2	
Incision and drainage of floor of frontal	1	
Miscellaneous surgery (4 operations—cured by use of tantalum foil; reported by Goodale in this case)	1	

TABLE 6.—NON-OSTEOMELITIC CASES.

		CASES	MORTALITY CASES
Total number of patients		176	19
No secondary surgery during follow-up		124	14
Patients having secondary frontal surgery		52	5
Total operations in these 52 cases:			
Nasal, sinus, cranial, etc.	224		
Directly on frontal sinus or sinuses	134		
Bilateral frontal at first operation	2		
One side, with other side later	8		
Obliterations		23	5
Further frontal surgery required	8		

TABLE 7.—OBLITERATION IN NON-OSTEOMYELITIC CASES

	CASES	FURTHER SURGERY CASES	MORTALITY CASES
Total patients	23		5
Type of surgery:			
Original surgical obliteration with removal of anterior wall and floor	6	6	
Lynch or other frontal surgery, then obliteration	17	2	

TABLE 8.—NON-OBLITERATION IN NON-OSTEOMYELITIS CASES WITH MULTIPLE OPERATIONS.

		CASES	MORTALITY CASES
Total patients		29	0
Total operations in these 29 cases	77		
Type of surgery:			
2 to 9 Lynch type operations		11	0
Miscellaneous frontal surgery followed by Lynch, or vice versa		16	0
Miscellaneous frontal surgery		2	0

TABLE 9.—SINGLE OPERATION ON ONE OR BOTH FRONTALS
(NON-OSTEOMYELITIC GROUP)

	CASES	MORTALITY CASES
Total patients	124	14
Type of surgery:		
Lynch	86	7
(31 were bilateral)		
Miscellaneous	15	2
(2 were bilateral)		
Obliteration	23	5
Technique:		
Removal of anterior wall and floor (one was bilateral)	17	0
Removal of all walls (two were bilateral)	5	5
All walls on one side; anterior wall and floor on other side	1	0

REFERENCES

1. Goodale, R. L.: Some Causes for Failure in Frontal Sinus Surgery, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:648 (Sept.) 1942.

2. Mosher, H. P.: The Surgical Anatomy of the Ethmoidal Labyrinth, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 38:869 (Dec.) 1929.

3. Knowlton, C. D., and McGregor, G. W.: How and When the Mucous Membrane of the Maxillary Sinus Regenerates, Arch. Otolaryng. 8:647, 1928.

4. Coates, G. M., and Ersner, M. S.: Regeneration of the Mucous Membrane of the Frontal Sinus after Its Surgical Removal (in a Dog), Arch. Otolaryng. 12:642, 1930.

5. Gorham, C. B., and Bacher, J. A.: Regeneration of Human Maxillary Antral Lining, Arch. Otolaryng. 11:763, 1930.

6. Arey, L. B.: Wound Healing, Physiol. Rev. 16:327 (July) 1936.

7. Howes, E. L.: The Rate and Nature of Epithelization in Wounds with Loss of Substance, Surg., Gynec. and Obst. 76:745, 1943.

8. Wolbach, S. Burt: Personal Communication, 1946.

9. Reifenstein, E. D., and Albright, F.: Paget's Disease; Its Pathologic Physiology and the Importance of This in Complications Arising from Fracture and Immobilization, New England J. Med. 231:343 (Sept.) 1944.

10. Boyd, W.: Pathology, Philadelphia, Lea and Febiger, 1941.

11. Lynch, R. C.: The Technique of Radical Frontal Sinus Operation Which Has Given Me the Best Results, Tr. Am. Laryng., Rhin. and Otol. Soc., 1920.

12. Sewall, E. C.: External Operation on the Ethmosphenoid-frontal Group of Sinuses under Local Anesthesia, Arch. Otolaryng. 4:377 (Nov.) 1926.

13. Smith, Ferris: Management of Chronic Sinus Disease, Arch. Otolaryng. 19:157 (Feb.) 1934.

14. Goodale, R. L.: The Use of Tantalum in Radical Frontal Sinus Surgery, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 54:757 (Dec.) 1945.

15. Kazanjian, V. H., and Holmes, E. M.: Reconstruction after Radical Oepration for Osteomyelitis of the Frontal Bone, Surg., Gynec. and Obst. 79:397 (Oct.) 1944.

16. Ingraham, F. D., Bailey, O. T., and Cobb, C. A.: The Use of Fibrin Film as a Dural Substitute, J. A. M. A. 128:1088 (Aug.) 1945.

17. Doherty, W. B.: Orbital Implaints, Arch. Ophth. 25:637, 1941.

18. Carlusci, G. A.: Am. J. Surg. 60:209, 1943.

19. Harmon, P. H.: Arthroplasty of the Hip for Osteoarthritis Utilizing Foreign-body cups of Plastic, Surg., Gynec. and Obst. 76:327, 1943.

20. Brown, J. M.: Frontal Sinusitis, Laryngoscope 56:116 (March) 1946.

21. Walsh, T. E.: Experimental Surgery of the Frontal Sinus. The Role of the Ostium and Nasofrontal Duct in Postoperative Healing, Laryngoscope 53:75 (Feb.) 1943.

22. Monto, R. W., and Lyle, P. L.: Treatment of Complicated Acute Sinusitis with Penicillin, Arch. Otolaryng. 43:298 (March) 1946.

23. Allman, C. H.: Personal Communication.

XXXIII

TINNITUS AURIUM: OBSERVATIONS ON THE EFFECT OF CURARE ON LOUDNESS LEVEL

MILES ATKINSON, M.D.

NEW YORK, N. Y.

In a recent paper on the nature of tinnitus,[1] an analogy was drawn between this symptom and the paresthesia which occurs with a neuritis or a neuropathy of peripheral nerves of common sensation. It was suggested that tinnitus aurium may be regarded as paresthesia of the auditory nerve and that its cause may well be a vascular disturbance such as has been suggested for other peripheral sensory neuropathies. Experiments were described showing the effect on the symptom of vasoconstrictor and vasodilator drugs and the results of the treatment of a series of cases with nicotinic acid were given.

In discussing this matter with a colleague, the question was raised as to how far tinnitus was psychogenic and the result of tension in the intrinsic muscles of the ear, and how far the success of treatment consequently was due to a psychological factor. That emotional disturbance has a part in the production of tinnitus in some cases is not to be denied, and the idea came to mind that some light might be shed on the role played in the production of tinnitus by muscular tension by using the drug curare to paralyze temporarily the intrinsic muscles of the ear. This paper describes the results of such an investigation.

Curare acts specifically on striped muscles by interrupting the nervous impulse at the termination of the nerve fibers in the muscles themselves. This action is the result probably of a neutralization of the acetylcholine reaction, which constitutes the fundamental neuromuscular mechanism.

When a drug having a pure curare action is introduced intravenously, it rapidly produces a paralysis involving all the skeletal muscles, the degree of paralysis being directly proportional to the amount of the drug given. The diaphragm and the intercostal muscles are the last to be affected, and embarrassment of respiration is therefore a late phenomenon. The drug is excreted rapidly so that the peak effect exists for only a few minutes and the duration of maximum response is transient.

As regards the ear, the two intrinsic muscles, the stapedius and the tensor tympani, are both striped muscles. The stapedius, the smallest striped muscle in the body, is supplied by a branch of the seventh nerve; while the tensor tympani, which belongs phylogenetically to the jaw musculature, is supplied by the motor root of the fifth nerve through the otic ganglion. Thus both their histological structure and their nerve supply put these two into the category of voluntary muscles. Their mode of action confirms this.

The tensor tympani muscle by its normal tone holds the malleus and the tympanic membrane in such a position that sound vibrations of moderate degree just cause movement of the incus and the stapes. When a sound of special import to the individual is heard, his name for instance, his attention is aroused and there is some evidence to show that this makes the tensor tympani contract, bringing the malleus and the tympanic membrane inwards into the optimum position for sounds to be carried as vibrations to the cochlea.[2] This action is comparable to the reflex ear twitch of animals. In man actual voluntary contraction of the tensor has been observed.[3] The stapedius muscle by its normal tone pulls on the stapes in such a manner that the endolymph in the cochlea is kept under slight pressure. When the muscle contracts, this pressure is increased and hearing becomes more acute. This action is comparable to cocking of the ear in animals in order to locate and intensify sounds. Intensive listening in man involves a similar action, a turning of the head in the direction of the sound and an effort to hear which is quite evident to the listener. It is highly probable that such intense listening in man involves an actual voluntary contraction of the stapedius.

From the above it follows that these two muscles, being both voluntary striped muscles, should be susceptible to the action of curare, and it is on the assumption of such an action that the rationale of the present investigation depends. If tinnitus is the result, in part or in whole or even in some cases only, of an increased tension of one or both of these muscles, then paralysis of them with curare should diminish temporarily its intensity.

The preparation of curare used was Intocostrin (Squibb). A 1 mg./kilo dose was injected in two parts; there was given at first the total dose less 20 mg. for safety's sake. The needle was kept in the vein and if, by the time the general musculature was relaxed, as shown by drooping of the eyelids and difficulty in raising the arm, no appreciable effect had been obtained on the level of the tinnitus and no respiratory embarrassment was evident, the final 20 mg. were injected. This was usually two to four minutes later.

Before the injection it was explained to the patient exactly what he might expect as regards muscular relaxation, and that he need have no fear because the effect was purely temporary. Then he was asked to note the existing level of the tinnitus and to observe carefully as the injection took effect whether this level changed, whether it became worse, better, or remained the same. Stress was always laid on worse rather than better. In addition, as a matter of interest, an audiometer reading at 1024 c. p. s. was taken before the injection was made and at four minute intervals thereafter for three readings. No attempt was made to balance the loudness level of the tinnitus with the audiometer, however, because of the transient nature of the curare action and the difficulty which many patients find in assessing this level at all accurately, even under the best of circumstances. The results of such attempts at balancing, because expressed in frequencies and decibels, tend to be invested with a scientific accuracy which they do not deserve. They are in fact just as much subjective as the statement of the patient that his noises have become louder or softer—indeed the statement may be more accurate than the measurement since the patient has no extraneous factor such as bell push and audiometer to distract his attention. For these reasons, in this investigation reliance has been placed upon the patient's statement with, as explained, stress laid upon the bad things to be expected rather than upon the good.

Certain precautions were always taken. An assistant was always present, ostensibly to take notes but more importantly in case of respiratory embarrassment in order to help with artificial respiration should this be necessary. In actual fact, it never was. Oxygen was always available together with a syringe containing 1 cc. of prostigmin which is the physiological antidote to curare. Neither of these was ever used; and in only one case did any difficulty in breathing appear, and this was of minor degree and short lived.

The greater part of the effect of the drug wears off within 10 minutes, but there remains for 30 minutes or more some difficulty in visual accommodation and a sense of unsteadiness. The complete muscular relaxation at the peak of the action is not an unpleasant sensation; indeed one particularly tense patient (Case 1) said that he had not felt so pleasantly relaxed for years.

Fifteen cases in which tinnitus was a predominant symptom were investigated. Ten were men, 5 were women. Their ages ranged from 27 years to 52 years. The duration of the tinnitus ranged from 2 years to 17 years with the exception of 1 case, No. 6, in which the tinnitus had been present for two weeks only. Tinnitus was

intermittent in 3 cases, though in all 3 was, of course, present at the time of the investigation. In the remainder it was constant, and in 6 cases severe. Hearing loss was present in all except 1 (Case 6). Of the 14 cases of deafness, 8 were of the perceptive type, 4 were of the conductive type and 2 were mixed. Four were cases of Ménière's syndrome.

RESULTS

Reduction of Tinnitus. In 4 cases (Nos. 1, 7, 8, 13) the level of loudness of the tinnitus was markedly reduced though in no case was the symptom entirely abolished. In all 4 cases the tinnitus had been constant and in 2 of them it was severe. The type of deafness was perceptive in 2 cases, conductive in 1, and mixed in 1. Two of them were cases of Ménière's syndrome. In two of these cases (Nos. 1 and 8), there was a marked psychoneurotic element while Case 13 had been under severe emotional strain for some months and her tinnitus, which previously as the result of treatment with nicotinic acid had become very mild, had lately increased in intensity and become very troublesome. In the fourth case (No. 7), the tinnitus was described as being of two kinds, "drumming" and "rushing water." The drumming was completely abolished during the action of the drug, the rushing water sound was diminished. This was a case of Ménière's syndrome in a married woman whose intellectual endowment was so poor that it was impossible to obtain an accurate audiogram. It is difficult to imagine this patient as tense in any sense of the word, and although she admitted having an extra-marital relationship with a married man several years her senior, there appeared to be no emotional backlash to this. She was, however, very definite about the abolition of the "drumming" tinnitus. It is possible that this sound was produced by tetanic contractions of one or both of the intrinsic muscles.

Increase in Tinnitus. In 2 cases (Nos. 6 and 9) the tinnitus was increased. Case 6 was the one already mentioned in which there was no associated deafness and the tinnitus ceased spontaneously after three weeks. The cause of the tinnitus was never definitely established but it was probably psychogenic, although the possibility of a psychic factor was strenuously, perhaps too strenuously, denied by the patient, a student nurse. Nevertheless, it seems more than possible that despite her denial there was an element of emotional strain in it, for when first seen she was about to take her final examination and had an air of strain about her, while when seen later after the examination she was smiling and happy and her tinnitus had

ceased. In Case 9, the tinnitus was subsequently abolished after treatment with nicotinic acid over a period of several months. In both cases the degree of worsening was said to be slight and very transient. It is difficult to find an explanation for this result in these two cases, unless it was due to an overstressing before the injection of the possibility of worsening.

No Change in Tinnitus. In 8 cases no change in the level of the tinnitus was noticed by the patient. Two of these were cases of Ménière's syndrome, Cases 10 and 11; one patient (Case 15) had otosclerosis and tinnitus of severe degree and a fenestration operation had previously been performed upon the ear tested. In 2 of the cases, (Nos. 5 and 12) a personality factor was also present. Case 5 had been discharged from the Army for combat fatigue; Case 12 was the subject of a severe emotional disturbance which had been present for the whole of the 16 years during which she had suffered from tinnitus. In the first of these patients, there was a definite hearing loss of conductive type which would not be related to the emotional factor; in the second, the emotional disturbance was severe enough to be called a mild psychosis and will be discussed later.

Change in Hearing Level. In 3 cases (Nos. 1, 10 and 13) there was a change in hearing level following administration of the drug—in 2 cases an improvement of five decibels and in 1 of ten decibels. In Cases 1 and 13, with five decibels improvement, the change in the hearing occurred coincidentally with the improvement in tinnitus. Case 10 which showed a 10 decibel improvement in hearing level was a case of Ménière's syndrome in which no improvement in tinnitus occurred under the influence of the drug. Here again an explanation is difficult to find. If the function of these muscles is accepted to be that described above, any change in hearing by paralysis of the muscles should be for the worse.

DISCUSSION

The object of this investigation was to discover what effect paralysis of the intrinsic muscles of the ear induced by curare might have upon the loudness level of tinnitus. Because emotional states are associated with increased muscular tension, certain cases in which a psychogenic element was present and therefore might play a part in the production of the tinnitus were deliberately selected for investigation. The remaining cases were all examples of apparently straightforward chronic progressive deafness with or without vertigo.

From the psychosomatic viewpoint the cases may be divided into three groups: (1) cases of chronic progressive deafness or of Ménière's

syndrome without any evidence of psychological disturbance; (2) cases in which an emotional element was very apparent; (3) a single case in which the psychological disturbance was of sufficient degree to be called a mild psychosis.

Group 1—cases without psychological involvement—comprises 10 cases. In one of these (Case 7), some improvement was manifested after the injection of the drug. In 2, a very slight worsening was obtained. In the remaining 7 there was no change.

Group 2 consists of 4 cases in which a definite psychogenic element was present. Three of these 4 experienced improvement in tinnitus after injection of the drug while 1 reported no change.

Group 3 consists of one case only. This case has been separated from the others because, as already stated, the patient was the subject of a mild psychosis. The noises of which she complained were not in the strict sense of the term tinnitus; they were described as being in the head, not in the ears, and were more in the nature of an auditory hallucination. The case is perhaps of sufficient interest to report briefly.

The patient was a married woman, aged 40 years, who, some 16 years previously, developed ringworm of the hands. This condition had proved very resistant to treatment of all sorts and had been a source of great trouble to her in performing her household duties because she had been forced to wear gloves whenever she had to put her hands in water. About the same time as the ringworm, the noises started and the patient definitely related the one event to the other. She exhibited marked paranoid trends, particularly with regard to members of her family, spoke much of her nerves and with regard to the noises said that at one time she was able to control them but now they controlled her. The noises were all over the head, and at times became very loud, almost like voices. She also complained of hearing loss in both ears which had come on gradually over the past ten years. The degree of this deafness was difficult to assess. There seemed to be some definite hearing loss, but responses to individual frequencies on the audiometer varied greatly from one test to another. When tested with a tuning fork (C.—512) she claimed a total loss of hearing by air conduction in both ears though she admitted to some degree of hearing by bone conduction, yet with this her response to conversational voice was not greatly impaired. A full dose of curare made no change in the noises. If they were in the nature of an hallucinosis as appeared probable, then of course none would be expected.

Thus, 3 out of the 4 cases which were benefited by curare were patients in which a psychogenic element was also present. Nine of 10 cases of emotionally uncomplicated chronic progressive deafness and tinnitus, 3 with vertigo, obtained no improvement, 2 of the 9 being made worse. These observations suggest, therefore, that spasm of the intrinsic muscles of the ear is not a factor in producing tinnitus except in cases in which a psychogenic element is also present.

These observations throw some light upon the unsatisfactory results of operations on the tensor tympani muscle. As long ago as 1847 Hyrtl suggested division of the tendon of the tensor tympani as a possible means of relieving tinnitus and of improving deafness. The operation was first performed on the living subject by Weber-Liel in 1868 and following this demonstration of its feasibility it had a vogue for a number of years with some otologists. Politzer, however, in the fourth edition of his textbook in 1902,[4] says of it: "According to the unanimous opinion of [various authorities], we may regard diminution in the subjective noises as an initial effect of tenotomy. The majority of the cases reported, however, cannot be used as examples in judging the effect produced by the tenotomy, owing to the failure to observe what effect incision of the membrane and the plicotomy alone had upon the subjective noises before the tendon of the tensor had been severed. The influence of tenotomy on the power of hearing is considerably less; a striking beneficial effect has seldom been observed. . . . There are even instances in which a decided change for the worse has been noted after the operation. . . . That this operation is of little value is evident when we consider that tenotomy, which was at first highly recommended by many authorities, has, of late years, been almost completely laid aside and practised only in exceptional cases."

In the 15 cases reported here, curare has been used to produce, by virtue of its paralyzing effect upon striated muscle, a physiological tenotomy not only of the tensor tympani but also of the stapedius muscle. The results obtained are largely in accord with those described by Politzer for surgical tenotomy.

SUMMARY

1. An investigation has been undertaken on 15 subjects in whom tinnitus was a predominant symptom, to determine the effect upon it of paralyzing the intrinsic muscles of the ear with the drug curare.

2. The method and dosage employed is described.

3. The results indicate that increased tension in the intrinsic muscles of the ear is not a factor in the production of tinnitus except in cases where an emotional disturbance is also present.

My thanks are due to Dr. Tibor Agoston for criticism and suggestions.

310 LEXINGTON AVENUE.

REFERENCES

1. Atkinson, M.: Tinnitus Aurium: Observations on its Nature and Control, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:742 (Dec.) 1944.

2. Yates, A. L.: The Principles and Practice of Otology (with F. W. Watkyn-Thomas), London, H. K. Lewis and Co., Ltd., 1932, p. 77.

3. Krieg, W. J. S.: Functional Neuroanatomy, Philadelphia, The Blakiston Company, 1942, p. 153.

4. Politzer, A.: Disease of the Ear, 4th ed., Philadelphia, Lea Brothers & Co., 1902, p. 307.

XXXIV

THE EXTERNAL EAR AND DRUM MEMBRANE IN OTOSCLEROSIS

FRANK WOJNIAK, M.D.

CHICAGO, ILL.

Many years ago someone defined otosclerosis as "a disease in which the physician sees nothing and the patient hears nothing". While not entirely true, this definition is an indication of the meager physical signs and symptoms in otosclerosis. Very seldom does the external auditory meatus or the tympanic membrane present any visible variations from the normal. On the other hand, there have appeared in the medical literature from time to time in the last eight or nine decades descriptions of physical signs and symptoms, among which may be mentioned lack of cerumen, enlargement of the lumen of the external auditory canal, atrophy of the skin of the canal, loss of its sensitivity or the tickling reflex, loss of the cough reflex, loss of the vascular reflex, Schwartze's sign, abnormal thinness and transparency of the drum membrane, increased mobility of the pars tensa of the drum membrane and reduced or absent mobility of the handle of the malleus. Such a well-recognized authority on the subject of otosclerosis as Holmgren,[1] in a recent contribution, stresses these signs and offers a highly speculative explanation. He suggests that an increased intralabyrinthine perilymph pressure, by causing pressure on the fenestra, results in partial immobility of the stapes, followed by gradual ankylosis. Secondary to this fixation the entire chain of ossicles becomes rigid. When the handle of the malleus is immobile, the nutrition of the tympanic membrane and of the walls of the external meatus is changed either mechanically or reflexly, with atrophy, decreased vascular reflex, increased transparency, decreased irritability and sensitivity, and reduced production of cerumen as consequences. This hypothesis, according to Holmgren, places all the symptoms of the external and the middle ear in relation one with another, and explains their connection with the basic condition.

A careful comparative study of the external auditory meatus and the tympanic membrane was carried out in 100 cases of otoscler-

---

Presented before the Chicago Laryngological and Otological Society, Dec. 3, 1945.

osis and 100 controls. These patients were all from the office practice of another otolaryngologist. The diagnosis of otosclerosis was based on the history of progressive deafness of insidious onset without a preceding or accompanying otitis media, and the finding of a conduction form of hearing impairment with intact and essentially normal tympanic membranes and patent eustachian tubes.

The "controls" comprised 70 patients with normal hearing and 30 patients with forms of deafness other than otosclerosis. These latter consisted mainly of cases of nerve deafness; cases of deafness due to pathological changes in the middle ear or in the eustachian tubes were not considered suitable for this investigation.

Of the 100 cases diagnosed as otosclerosis, operations were done in 53. A satisfactory view of the stapes footplate through the binocular dissecting microscope was obtained in 32 of the operated cases. In every one of these, bony ankylosis of the stapes to the margin of the oval window was observed, and in some cases the entire stapes footplate was replaced by solid bone continuous with the margins of the oval window.

The following signs were studied in each case: (1) the size of the external auditory canal; (2) cerumen; (3) the condition of the skin in the external canal; (4) its sensitivity; (5) the cough reflex; (6) the vascular reflex; (7) the condition of the drum membrane with special reference to its thinness and transparency, Schwartze's sign, and the mobility of the handle of the malleus and the pars tensa.

The Size of the External Auditory Meatus. Gourivaud[2] in 1890 noted that in what he calls the sclerotic form of otosclerosis the auditory meatus is large, straight, and dry. Tweedie[3] in 1908 stated that the external canal in otosclerosis is often unusually patent, white, shining and deficient in wax. Politzer[4] stated in his textbook that in a number of cases of otosclerosis the meatus was narrowed through hypertrophy, whereby it could be concluded that there was diffuse hyperostosis of the temporal bone.

Bezold[5] gives the diameters of the meatus as follows: At the commencement of the cartilaginous part the greatest diameter is 9.1 mm., the least 6.5 mm.; at its end, the greatest diameter is 7.8 mm., the least 6.0 mm.; at the commencement of the osseous part, the greatest diameter is 8.7 mm., the least 6.1 mm.; at its end, the greatest diameter is 8.1 mm., the least 4.6 mm. The cartilaginous canal is narrower in children than in adults. In old persons a slit-like closure of the external orifice of the ear often takes place as a result of atrophy and shrinking of the cartilage.

The volume of the meatus of an adult is about one cubic centimeter according to Hummel.

To our knowledge there is no practicable method of measuring the lumen of the canal in the living with any degree of accuracy, but bearing in mind the approximate measurements as given by Bezold as well as the approximate volume of 1 cc. as given by Hummel, one is enabled by careful inspection to form a fair judgment as to its size.

Of the 100 patients with otosclerosis in this study, 41 had canals of normal size; 28 canals were slightly enlarged, 28 were moderately enlarged, 1 rather markedly enlarged, and 2 were definitely narrower than normal.

Of the 70 normal ears, 49 canals were of normal size, 10 slightly enlarged, 9 moderately so, none markedly, and 2 were quite narrow.

Of the 30 patients with deafness other than that due to otosclerosis, 12 canals were of normal size, 7 slightly enlarged, 8 moderately so, 1 markedly, and 2 were quite narrow.

These figures would seem to indicate that there is a slight tendency to enlargement of the external auditory canal in patients suffering from otosclerosis, but the same slight tendency was observed in cases of deafness from other causes, and it seems likely that the statistical differences between the deaf and the sound ears are within the normal limits of variation.

Cerumen. In 1857 Toynbee[6] observed that the external meatus is usually devoid of cerumen in far advanced cases of otosclerosis. Erhard,[7] Keene,[8] Richey,[9] and Politzer[4] observed that the external auditory canal is generally free from secretion, dry, and pale. Fraser and Walker[10] reported that of 26 cases the canal was wide and free from wax in 14, while in 12, wax was present. Leeson[11] found the auditory canal free from cerumen in 783 out of 912 cases of otosclerosis. In 1931 the British otosclerosis committee found the cerumen absent in 54 per cent of cases, from answers to a questionaire.

In estimating the amount of cerumen it was thought convenient to make use of the following terms: "absent"; "trace", when the cotton on an applicator showed a slight amount; "moderate", when the amount was sufficient to require two or three wipings in order to clear the canal; "considerable", when still more; and "impacted", when douching was necessary.

Of the 100 patients with otosclerosis in this study, there were only 6 with cerumen entirely "absent"; 61 with a "trace" of cerumen;

26 with a "moderate" amount; 4 with "considerable"; and 2 had "impacted" cerumen in either one or both canals.

Of the 70 normal ears, the cerumen was "absent" in 5; a "trace" was present in 41; "moderate" amount in 22; "considerable" in none; and "impacted" in 2.

Of the 30 cases of deafness other than that of otosclerosis, the cerumen was "absent" in 6 ears; a "trace" was present in 19; "moderate" amount in 4; "considerable" in none and "impacted" in 1.

The amount of cerumen appears to be the same in cases of otosclerosis, normal hearing, or impaired hearing from other causes. The patients' ages seemed to play a conspicuous role in the amount of cerumen, the younger subjects, in general, having more cerumen than the older ones.

Condition of the Skin in the External Canal. Politzer[4] states that the lining of the external auditory meatus is much stronger in the cartilaginous than in the osseous portion; in the latter the skin gradually becomes more delicate and thinner as it approaches the sulcus of the tympanic membrane. The dermic layer of the cartilaginous portion, one to two mm. in thickness, has an abundant growth of hair, into the sacs of which sacculated sebaceous glands discharge. We also find ceruminous glands in the deeper layers of the cutis which are more or less compact. The glandular layer extends from the posterior superior wall of the cartilaginous portion into the osseous meatus in the form of a triangle several millimeters long. The glandular elements are wanting in the other parts of the osseous meatus; the more delicate cutis, firmly united with the periosteum, forms compact spirally arranged folds and contains only a few papillae.

It has been claimed by some authors that there is an atrophy of the skin of the canal in patients with otosclerosis. In this study the condition of the skin was judged by such criteria as the color, apparent thickness, gloss and desquamation. It was considered normal when the color was fleshy-pink, when there was the impression of appreciable thickness, a gloss or sheen indicative of normal action of its glands, and no undue desquamation. It was considered atrophic when it seemed to be unusually pale or white in color, very thin, dull and dry.

Of the 100 cases of otosclerosis in this study the skin of the external canal was considered atrophic in 49. Of the 70 normal cases, in only 9 were there indications of atrophy, while of the 30 cases of other forms of deafness, 15 canals showed atrophy. Here again,

the age of the patient, and not his disease, seemed to be the determining factor; the younger the patient, the less atrophy of the skin. The average age of our normal controls is lower than that of the patients with otosclerosis, which seems to explain sufficiently the lower incidence of skin atrophy in the former.

The Sensitivity of the Skin of the External Canal and of the Tympanic Membrane. In 1858 Erhard[7] observed that the auditory meatus becomes anesthetic and leukomatous in cases of otosclerosis, while Richey[9] in 1886 noted that it is often irritable.

Itard and Cholewa[12] described anesthesia of the tympanic membrane in otosclerosis and said it is often associated with a diminished tactile sensation on tragus pressure and with the entrance of air into the middle ear on catheterization. This symptom, they stated, is encountered also in hysteria and neurasthenia, and that not improbably it is related to dryness and desquamation of the skin in the auditory canal.

In 1888 Cholewa stated that in otosclerosis there is a neuropathic element in the fifth and the sympathetic nerves, as shown by the anesthesia of the tympanic membrane. He thought it was an important diagnostic sign. In 1895 Zwaardemaker[13] spoke about a new sign of otosclerosis in the form of tactile insensibility of the tympanic membrane for massive air movements and air displacements.

Fröschels[14] in 1910 described a symptom that he found to be characteristic of otosclerosis. On careful examination of patients in the Vienna Ear Clinic, he noted that individuals with otosclerosis were much more tolerant of delicate manipulation in the external ear than were other patients. Systematic testing of over 300 patients of all kinds and 100 individuals with normal ears actually showed that a diminution of the tickling sensation can be demonstrated in three groups of affections, namely, in chronic suppurative otitis media, in typical otosclerosis (40 cases) and in a large number of cases suspected of otosclerosis. The typical cases of otosclerosis presented this symptom in nearly 93 per cent of cases.

Fröschels arrived at the conclusion that typical otosclerosis leads to a progressive diminution of the tickling sensation corresponding to the aggravation of the deafness. The author also found almost complete lack of reaction to tickling in 110 out of 120 congenital deaf-mutes, but the results permitted no conclusions as to the relation between congenital deafness and otosclerosis. The external auditory meatus is as a rule extremely ticklish, the posterior and the lower walls being the most sensitive.

Four nerves participate in the supply of the external ear: the fifth, the seventh, the tenth, and the cervical plexus. It is not known which of these nerves conducts the tickling sensation, but although the vagus undoubtedly plays a part in sensation in the external auditory meatus, the principal part is evidently played by the trigeminus; this nerve, therefore, according to Fröschels, is affected in otosclerosis, but it is not clear just how. As the nerves of the external auditory canal do not reach the middle ear, nor do the nerves from the middle ear pass into the external canal, and since these two parts of the ear are supplied by different branches of the nerve, the damage incurred by the fifth nerve in the middle ear cannot explain its failure in the external canal.

Beck[15] in 1910 found this symptom in 98 per cent of otosclerosis cases, and a suggestion of it in the remainder. In 1814 Fraser and Walker[10] reported tickling diminished in every one of their 138 cases and markedly so in the worse ear. They state, however, that this sign has been found in other forms of deafness, and that its value as a diagnostic sign is not very great.

In this study, in order to determine the sensitivity of the external ear canal, a cotton-tipped applicator was used. A variable amount of friction was applied, ranging all the way from barely touching the skin, to fairly moderate pressure. It was soon discovered that there is a marked variation in the sensitivity of the various portions of the canal, the posterior and inferior walls being distinctly more sensitive than the anterior and superior. Also, the sensitivity rapidly increases as one approaches the innermost portion of the canal, the drum membrane being the most sensitive.

To estimate the degree of sensitivity a definite technic was followed. The outermost portion of the canal was carefully swabbed in order to clean it of cerumen, and the patient's reaction watched. If no objection was registered, the walls of the canal were touched with the cotton using a spiral motion inward until the drum membrane was reached. In no instance have we found complete anesthesia. The response was labeled as "slight", when the patient failed to volunteer a complaint, but on questioning, admitted discomfort; "moderate", whenever the patient objected to the maneuver either verbally or by facial expression; "marked", when the patient jerked the head away. In the vast majority the two ears reacted equally; in a small number (14) the response was unequal. However, we were not able to foretell by this test alone the degree of hearing impairment in a given ear. Frequently, the response was different in the two ears of a patient with both ears perfectly normal.

Of the 100 patients with otosclerosis, 78 showed "slight" response to the sensitivity test, 19 "moderate", and 3 "marked".

Of the 70 normal controls, 49 showed "slight" response, 17 "moderate", and 4 "marked".

Of the 30 patients with other forms of deafness, 27 displayed "slight" response, 3 "moderate", while there were no "marked" responses.

These figures seem to indicate that the skin sensitivity test or the so-called "tickling reflex" is of no practical value in the diagnosis of otosclerosis.

Cough Reflex. At the same time that the sensitivity of the external canal was being determined, another symptom, the cough reflex, was watched for. This is a reflex phenomenon induced by irritation of the terminal filaments of the auricular branch of the vagus (Arnold's nerve). The introduction of the ear speculum or, more frequently, manipulation of the cotton-tipped applicator within the ear canal, often causes an annoying spasmodic "ear cough". This has been said to be markedly reduced or virtually absent in patients with otosclerosis.[1] Our study shows that this reflex has been elicited in 6 cases, while it was absent in 94 cases of otosclerosis. It was found to be present in 4 of the 70 normal patients, and in 2 of the 30 patients with other forms of deafness.

Condition of the Drum Membrane. According to Bezold,[5] the color of the drum membrane is influenced by its transparency. Normally, it is pearly gray. Towards the center it has a yellowish tint from the bone of the promontory which lies close behind it. The thinnest part of the drum membrane, the intermediary zone, appears diffusely pinkish, whenever the blood vessels of the inner wall are injected. The thicker and therefore opaque parts of the membrane are more whitish.

Toynbee[6] in 1857 stated that the tympanic membrane in otosclerosis is often more opaque than normal; sometimes it shows patches of opacity, and is occasionally externally more concave than normal. In 1858 Erhard[7] stated that the drum membrane becomes opaque and disc-like in consequence of the thickening of its posterior fold. Keene[8] in 1873 observed that the tympanic membrane may appear healthy or may show thickening of concentric or circular fibers and white radiating or crescentic patches. In advanced cases the membrane is tense and glittering. Ladreit de Lacharrière[16] in 1881 stated that the tympanic membrane may be normal or it may

be thickened and opaque. Richey[9] in 1886 noted a "general atrophy of the conducting mechanism of the ear." The drum membrane is blanched, often striated, and motionless during inflation.

Gourivaud[2] in 1890 stated that otosclerosis may be either hyperplastic or of the typical sclerotic form. The hyperplastic form is characterized by vascularization of the ossicles; there are new-formed vessels arranged in a special way; they are parallel to the handle of the hammer and when air is insufflated into the middle ear they become redder. There is also hyperemia of the promontory and the drum membrane is more indented than normal. In the sclerotic form the drum membrane is normal or thin, atrophied and transparent; in other cases it is yellow, opaque and streaked, with irregular sclerotic bands, and its mobility is decreased.

Axmacher[17] in 1902 stated that when the tympanic membrane is atrophied the handle of the malleus, which is enlarged, can often be seen in the form of whitish opacity.

Politzer[4] in his book states that in a great number of cases we find the tympanic membrane absolutely normal in appearance. It occasionally happens that the membrane is dull, intensely cloudy, atrophic, and sometimes retracted. Such conditions are seen especially in old individuals, and in those forms of catarrhal adhesive processes of the middle ear combined with sclerosis.

In 1903 Heiman[18] noted a prominence of the anterior fold of the tympanic membrane in 11.6 per cent of his cases of otosclerosis.

In 92 out of 113 cases in which the diagnosis of otosclerosis was made by Shambaugh and Holderman[19] the tympanic membrane was typically normal. In 21 cases the diagnosis of otosclerosis was made in the presence of alterations of the drum membrane that indicated "an exhausted tubotympanic process." Such alterations usually indicate a condition that develops in early childhood and rarely leaves any permanent defect in hearing. In such cases the diagnosis of otosclerosis was made only when the evidence of fixation of the stapes was out of proportion to the slight changes in the drum membrane, when there was no active tubal process, when there was a history of insidious onset of deafness and the occurrence of other cases of otosclerosis in the family.

A number of observations enter into the consideration of the condition of the drum membrane, such as its thinness or thickness, color, translucency or opaqueness, its vascularization, and the mobility of its various parts. The pars tensa of a thin drum membrane is

generally translucent, the long process of the incus frequently showing through. A thick drum membrane is generally opaque and dull. In this study an attempt was made to establish the degree of thinness of the drum membrane from the impression of its appearance and the degree of mobility of its pars tensa. The thinness was found to be "marked" in 8, "moderate" in 35, "slight" in 31, and "normal" in 26, while no distinct thickening of the drum membrane was found in any of the 100 cases of otosclerosis.

Of the 70 normal hearing patients, the thinness was "marked" in 1, "moderate" in 9, "slight" in 7, "normal" in 48 and in 5 there was thickening. Of the 30 patients with other forms of deafness, the thinness was "marked" in 3, "moderate" in 14, "slight" in 3 and "normal" in 10.

Vascular Reflex. The skin of the posterior and superior part of the bony meatus contains the blood vessels and the nerves of the drum membrane, enclosed in a layer of subcutaneous cellular tissue. The rest of the bony meatus is lined with thin cutis which is closely connected with the periosteum.

In 1890 Gourivaud[2] stated that in the hyperplastic form the blood vessels along the handle of the malleus become redder when air is insufflated into the middle ear.

Holmgren[1] states that if the inner end of the auditory canal and the drum membrane are stimulated mechanically by contact with a swab of cotton or by strong aspiration and compression with Siegle's otoscope a lively reflex hyperemia normally results in the tympanic membrane and particularly in the skin of the posterior upper auditory canal. In cases of otosclerosis this result will be entirely absent or reduced in numerous cases, according to Holmgren.

In our study we have found this reflex to be "marked" in 16, "moderate" in 34, "slight" in 47, and "absent" in 3 cases of otosclerosis.

Of the 70 normal hearing patients the vascular reflex was "marked" in 10, "moderate" in 39, "slight" in 20, "absent" in 1. Of the 30 patients with other forms of deafness it was "marked" in 5, "moderate" in 8, "slight" in 16, and "absent" in 1.

Schwartze's Sign. Denker[20] in 1904 stated that the translucent hyperemia of the mucosa of the promontory points with great probability to an existing inflammatory process in the labyrinthine capsule and at the stapediovestibular symphysis.

Politzer[4] in his book states that a reddish luster behind the umbo is an important, although inconstant symptom, which, according to the author's observations, is produced by a hyperemic condition of the blood vessels of the osseous wall of the promontory, generally accompanying otosclerosis. Sometimes cases are met with in which this reddish luster of the wall of the promontory is spread over the entire membrana tympani.

Bezold[5] states that an especially poor prognosis must be given in cases which continually show the transparent redness of the promontory. Lucae[21] in 1907 was in agreement with Politzer in interpreting the red reflex at the promontory as the expression of disease of the labyrinthine capsule, not of the tympanic mucosa—the congested bone vessels showing through the mucosa. Its absence denotes a more advanced process. Heine[22] observed that the red reflex may vary in the same patient in such a way as to be sometimes barely recognizable while at other times it is extremely well developed.

Shambaugh in 1908 stated that congestion of the mucous membrane covering the promontory may be constantly present or may be brought out only by catheterization of the eustachian tube. Of 113 cases in which the diagnosis of otosclerosis was made by Shambaugh and Holderman in 1926, in 15 a distinct pinkish glow was present on the promontory, a symptom recognized as pathognomonic of otosclerosis, but observed usually in the earlier stages of the process.

Tweedie[3] in 1908 stated that the drum membrane may be normal, nearly normal, or of a peculiar pinkish hazy appearance. Fifteen of 138 cases observed by Fraser and Walker[10] in 1914 showed a flush of the promontory. Sewell[23] stated in 1910 that Schwartze's sign is rare and that Siebenmann found it in nerve deafness. Alexander stated in 1912 that the promontorial blush is an important diagnostic adjuvant, but may be also found with a perfectly normal auditory organ.

Brühl[24] notes in 1913 that a transparent red sheen on the wall of the promontory may occur occasionally in a person with normal hearing. In 1925 Lake[25] stated that the red reflex demonstrates that a patch of rarefying osteitis exists underneath the pinkish areas of aural mucosa, and as such it is of interest. Drury[26] in 1926 stated that the reddish blush on the promontory seen through the ear drum was a nearly constant finding in his series.

We have observed in this study a distinct pinkish glow, most frequently posteriorly to the handle of the malleus, in 21 out of 100

cases of otosclerosis. This sign was also present in 3 of the 70 normal hearing patients, and in 3 of the 30 with other forms of deafness.

Mobility of the Handle of the Malleus and Pars Tensa. As far back as 1858 Voltolini[27] maintained that frequently, but by no means invariably, ankylosis of the stapes is associated with ankylosis of the other ossicles so that the tympanic membrane is immovable. From time to time numerous observers entered into considerable controversy. regarding this point.

Helmholtz[28] maintained that the free part of the tympanic membrane moves three to four times as much as the tip of the handle of the malleus. Movements of the tympanic membrane are transmitted by way of the auditory ossicles in such a manner that the movements of the stapes are considerably less than those of the handle of the malleus. With change of pressure the movement of the handle increases more and more in comparison with that of the incus, and still more in comparison with the stapes. It is generally pointed out that the inward-outward movement of the handle of the malleus is the most prominent and important, being greater with aspiration than with compression in the ratio of 5 to 3 (according to Dahman).

Holmgren[1] pointed out the errors in describing the tympanic picture and called attention to the necessity of trying to estimate the mobility of the handle of the malleus. As it is difficult to determine this with any degree of accuracy, he uses a Siegle otoscope in which two hairs are set parallel and fairly close to each other on either side of the center of the lumen. The speculum is held in the auditory canal in such a manner that the handle of the malleus lies between the two hairs. On compression and aspiration of air the movements of the handle can now be observed between the hairs.

Frenckner[29] stated that while the fixity of the stapes in otosclerosis is an established fact, the movements of the ossicles and the tympanic membrane are assumptions. In order to throw some light on this subject, he filmed the movements of the tympanic membrane and of the malleus during compression and aspiration of air in the outer auditory canal, and made careful calculations of the amplitude of the movements of the handle of the malleus. The pressure ranged between +35 mm. and —14 mm. of mercury. Higher pressure variations were unpleasant or painful, and more so with negative than with positive pressures. Frenckner claims that his studies justify the following conclusions: In cases of oto-

sclerosis, where the tympanic membrane appears to be fully normal, one meets with the most variable conditions. The mobility of the free part of the membrane, the pars tensa, is the same in the healthy ear and the otosclerotic ear, and stronger during aspiration than during compression. The movement of the handle of the malleus is, contrariwise, in some cases of otosclerosis, it is so inconsiderable that it cannot be discerned on a film greatly enlarged and, with mathematical calculations of drawings, only an exceedingly inconsiderable movement of the handle can be proved even with great difference of pressure. Again, in other cases, on membranes having the same normal appearance, one meets with handle movements which more or less approach the normal. Otosclerotic change in the malleo-incudal joint or in the neighborhood of its surfaces appears to the author to be a possible explanation of the fixity of the handle.

Covell[30] recently confirmed the observations of Holmgren and Frenckner. He went through Noger's large collection of histological specimens of otosclerosis and observed a distorted position of the ossicles in 95 per cent of cases with otosclerotic foci in the region of the stapedio-vestibular articulation. Covell considers the fixation of the stapes directly responsible for the rigidity in the other articulations.

In this study, to determine the movements of the tympanic membrane we made use of a Siegle otoscope in which two parallel hairs were placed, as recommended by Holmgren. Compressing the rubber bulb with approximately the same force in all cases, an attempt was made to estimate the excursions of the pars tensa and of the handle of the malleus. The extent of these excursions is designated as "marked", "moderate", "slight" or "absent".

The mobility of the handle of the malleus in 100 cases of otosclerosis was "marked" in none of the cases, "moderate" in 17, "slight" in 36, "absent" in 47 It was "marked" in 3 of the 70 normal hearing controls, "moderate" in 22, "slight" in 35, and "absent" in 10. It was "moderate" in 5, "slight" in 18, and "absent" in 7 of the 30 patients with other forms of deafness.

The mobility of the pars tensa in 100 patients with otosclerosis was "marked" in 85, "moderate" in 12, "slight" in 3. Of the 70 normal hearing patients, it was "marked" in 48, "moderate" in 19, "slight" in 3, while of the 30 cases of other forms of deafness it was "marked" in 19, "moderate" in 10, and "slight" in 1.

These figures would indicate that there is an apparent absence of mobility of the handle of the malleus in 47 per cent of patients with otosclerosis as compared with only 14 per cent of those with

normal hearing and 24 per cent of those suffering from deafness other than that due to otosclerosis. In another 36 per cent of patients with otosclerosis the mobility is so slight as to be barely discernible. The pars tensa of the drum membrane in patients with otosclerosis appears to be only moderately more movable than in those with normal hearing or other forms of deafness.

SUMMARY

Concrete evidence in the way of readily demonstrable physical signs and symptoms is difficult to obtain and, when obtained, its interpretation is dependent to a great extent on the individual examiner. Many of these signs, such as the atrophy of the skin or the thinness of the drum membrane, are of necessity merely visual impressions, and as such, are subject to wide variations with different observers. For this reason, significance of any one special physical sign or symptom, or even a combination of several of them, may be doubtful or misleading.

The data obtained in the course of our investigation can be briefly summarized as follows:

1. The size of the external auditory canal in patients with otosclerosis is generally larger than that of normal hearing subjects, but coincides very closely with that of patients suffering from other forms of deafness.

2. The amount of cerumen appears to bear no relation to deafness and normal hearing.

3. The skin in the external canal appears to be atrophic as often as it appears to be normal in patients with defective hearing regardless of the type of deafness. Only 12.8 per cent of the normal hearing patients showed signs of atrophy.

4. The sensitivity of the skin, or the so-called "tickling reflex", shows no appreciable variations between our series of patients with otosclerosis and the controls.

5. The cough reflex appears to be of no diagnostic significance. It was elicited as frequently in patients with otosclerosis as in those with other forms of deafness or those with normal hearing.

6. The drum membrane seems to be thinned out abnormally only slightly more frequently in otosclerosis than in other forms of deafness.

7. There is no marked variation in the vascular reflex of patients with otosclerosis and those with other forms of deafness; one could say, however, that in normal hearing subjects this sign is more readily elicited.

8. Schwartze's sign appears to be highly suggestive of otosclerosis, being present in 21 per cent of the patients with that disease. However, it was present in 10 per cent of those with other forms of deafness, and in 4.2 per cent of the normal hearing patients.

9. Mobility of the handle of the malleus appears to be absent in 47 per cent of patients with otosclerosis as compared with 23.3 per cent of those with other forms of deafness, and 14.2 per cent of those with normal hearing.

10. The difference in the mobility of the pars tensa of the drum membrane between the three groups studied is negligible.

CONCLUSIONS

It would appear from the present investigation that the external ear and the drum membrane have not a great deal to offer in the diagnosis of otosclerosis. Atrophy of the skin of the external auditory canal, the abnormal or unusual thinness of the drum membrane appear to be more frequent in cases of deafness than in normal hearing individuals, but no more frequent in otosclerosis than in other forms of deafness. Except for Schwartze's sign, which is present twice as often in otosclerosis as in other forms of deafness, and five times as often in otosclerosis as in normal hearing individuals, and the apparent absence of mobility of the handle of the malleus in a greater proportion of patients with otosclerosis than of the controls, there is nothing characteristic in the external ear and drum membrane in otosclerosis to help in its differentiation from other forms of deafness.

The author is grateful to Dr. G. E. Shambaugh, Jr., for his valuable suggestions in the preparation of this paper.

4649 SOUTH ASHLAND AVE.

TABLE 1.—EXTERNAL EAR IN OTOSCLEROSIS, OTHER FORMS OF DEAFNESS AND NORMAL HEARING.

			CONTROLS, 100 CASES	
		OTOSCLEROSIS 100 CASES	OTHER FORMS OF DEAFNESS 30 CASES	NORMAL HEARING 70 CASES
Size of External Auditory Canal	Narrower than normal	2%	6.6%	2.8%
	Normal	41%	40%	70%
	Slightly enlarged	28%	23.3%	14.2%
	Moderately enlarged	28%	26.5%	12.8%
	Markedly enlarged	1%	3.3%	0
Cerumen	Absent	6%	20%	7.1%
	Trace	61%	63.3%	58.5%
	Moderate	26%	13.3%	32.8%
	Considerable	4%	0	0
	Impacted	2%	3.3%	2.8%
Condition of the Skin	Normal	51%	50%	87.1%
	Atrophic	49%	50%	12.8%
Sensitivity of the Skin	Slight	78%	90%	70%
	Moderate	19%	10%	24.2%
	Marked	3%	0	5.7%
Cough Reflex	Present	6%	6.6%	5.7%
	Absent	94%	93.3%	94.2%
Thinness of Tympanic Membrane	Marked	8%	10%	1.4%
	Moderate	35%	46.6%	12.8%
	Slight	31%	10%	10%
	Normal	26%	33.3%	68.5%
	Thickened	0	0	7.1%
Vascular Reflex	Marked	16%	16.6%	14.2%
	Moderate	34%	26.6%	55.7%
	Slight	47%	53.3%	28.5%
	Absent	3%	3.3%	1.4%
Schwartze's Sign	Present	21%	10%	4.2%
	Absent	79%	90%	95.5%
Mobility of Handle of Malleus	Marked	0	0	4.2%
	Moderate	17%	16.6%	32.8%
	Slight	36%	60%	50%
	Absent	47%	23.3%	14.2%
Mobility of Pars Tensa	Marked	85%	63.3%	68.5%
	Moderate	12%	33.3%	27.1%
	Slight	3%	3.3%	4.2%
	Absent	0	0	0

REFERENCES

1. Holmgren, G.: Otosclerosis. Symptoms in the External and Middle Ear, Acta Oto-laryng. 29:91-97, 1941.

2. Gourivaud, A.: Contribution a l'etude de l'ankylose des osselets de l'ouïe, Toulouse, 1890.

3. Tweedie, A. R.: Otosclerosis; Some Points in its Etiology, Diagnosis, and treatment, Lancet 2:1808, 1908.

4. Politzer, A.: Diseases of the Ear, Philadelphia and New York, Lea Brothers & Co., 1903.

5. Bezold, F., and Siebenmann, F.: Otology, Chicago, E. H. Colegrove Co., 1908.

6. Toynbee, J.: Diseases of the Ear, 2nd Ed., 1865. Quoted by Politzer, Ref. 3.

7. Erhard: Ueber Ankylose des Steigbugels als Ursache der Schwerhörigkeit, Vrtljschr. f. d. prakt. Heilk. 57:148, 1858.

8. Keene, J.: The Causes and Treatment of Deafness, London, 1873.

9. Richey, S. O.: General Atrophy of the Conducting Apparatus of the Ear, Arch. Otol., 15:17, 1886.

10. Fraser, J. S., and Walker, G.: Clinical Aspects of Otosclerosis, J. Laryngol. 29:513, 1914.

11. Leeson, L. A.: The Clinical Aspects of Otosclerosis, J. Laryngol. & Otol. 43:89, 1928.

12. Cholewa, R.: Ueber progressiva Schwerhörigkeit und ihre Behandlung durch die Tenotomie des Tensor Tympani, Ztschr. f. Ohrenh. 19:240, 1888.

13. Zwaardemaker, H.: Ein Initial Symptoms der Sclerose, Ztschr. f. Ohrenh. 28:119, 1895.

14. Fröschels, E.: Ueber ein neues Symptom bei Otosklerose, Monatschr. f. Ohrenh. 44:23, 1910.

15. Beck, O.: Ueber die Bedeutung der Syphilis für die Pathologie der Otosklerose, Monatschr. f. Ohrenh. 44:536, 1910.

16. Ladreit de Lacharrière: Des engorgements et de l'ankylose des articulations des osselets de l'ouie, Ann. d. mal. de l'oreille, du larynx 7:133, 1881.

17. Axmacher, F.: Beitrag zur Behandlung der Sklerose der Pankenhöhle, Göttingen, University Thesis, 1902.

18. Heiman, T.: Die Otosklerose, Monatschr. f. Ohrenh. 43:761, 1909.

19. Shambaugh, G. E., and Holderman, J. W.: The Occurrence of Otoslerosis in the Etiology of Progressive Deafness, Arch. Otolaryng. 4:127, 1926.

20. Denker, A.: Die Otosklerose, Wiesbaden, (Ohrenheilkunde der Gegenwart, No. 4), 1904.

21. Lucae, A.: Die chronische progressive Schwerhörigkeit, Berlin, 1907.

22. Heine, B.: Ueber die sogenannte Otosklerose, Therap. d. Gegenwart. 53:21, 1912.

23. Sewell, D. L.: Otosclerosis, Practitioner 84:237, 1910.

24. Brühl, G.: Ueber Otosclerose, Berlin Klin. Wchnschr. 47:2300, 1910.

25. Lake, R.: Otosclerosis; Does it Exist as a Separate Disease? J. Laryngol. 40:512, 1925.

26. Drury, D. W.: Otosclerosis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 35:651, 1926.

27. Voltolini: quoted by P. Frenckner, Ref. 29.

28. Helmholtz: quoted by P. Frenckner, Ref. 29.

29. Frenckner, Paul: Movements of the Tympanic Membrane and of the Malleus in Normal Cases and in Cases of Otosclerosis, Acta Oto-laryng. 27:587, 1939.

30. Covell, W. P.: Pathology of Ossicles, Acta Oto-laryng. 28:263, 1938.

XXXV

TUNING FORK TESTS

A HISTORICAL REVIEW

KURT TSCHIASSNY, M.D.

CINCINNATI, OHIO

It may be of interest to anatomists as well as to otologists that just 400 years ago the stapes, the smallest bone in the human body, was discovered.

The credit of having first described it, in 1546, goes to Giovanni Filippo Ingrassia. Andreas Vesalius,[2] Ingrassia's teacher in anatomy had previously described four ossicles, two on each side. "Auditus organi ossicula quattuor sunt duo scilicet ad singulas aures." Ingrassia when he by chance discovered the third ossicle christened it "stapes" because, as he wrote, its resemblance to the stirrup is even greater than the similarity of the two other ones to the hammer and the anvil. "Quia longe majorem similitudinem hoc ossiculum habet cum stapede quam alia duo cum malleo et incude." There was for a short time an argument concerning the priority of this discovery between Ingrassia, Fallopio, Eustachio, Collado, and Colombo. But when Fallopio left in his honesty the priority to Ingrassia, the other ones also retired.

Ingrassia, however, deserves also the merit of another discovery which centuries later turned out to be of fundamental importance for clinical otology. He was the first one in medical history to realize the fact that a vibrating tuning fork can be heard when pressed against the teeth. Thus he was the first observer of bone conduction.

Biographical Note. Ingrassia (1510-1580) was Professor of Anatomy at the medical schools of Padua, Naples and finally in Palermo. He was one of the most famous teachers of his time and excelled himself during the plague epidemic in 1575. He was honored by the title: "Hippocrates Siculus."

"Ingrassia's phenomenon", generally called bone conduction, is the basic principle of our routinely used tuning fork tests. In this

---

From the Department of Otolaryngology, College of Medicine, University of Cincinnati.

connection a brief review of the history, the principles and the evaluation of these tests may be justified.

The well-known common goal of these tests is the determination of the site of the lesion in the auditory apparatus in order to differentiate between the conduction and the perception types of deafness. The common technical principle of these tests is the utilization of bone conduction, i.e., of sound waves produced by a vibrating tuning fork and transmitted by the skull bone to the ears. While thus the means of comparison is the same for the tests of Weber, Rinne, and Schwabach the things compared differ in each test.

Weber's Experiment: While it was in the sixteenth century that bone conduction was discovered, it was not before the nineteenth century that the various comparative experiments were performed, constituting the basis of our present tuning fork tests. The first of these experiments is generally credited to Ernst Heinrich Weber. His first studies on the transmission of sound and air waves, performed in collaboration with his brother Wilhelm, were published in the year 1825.[3] The exact description of Weber's observation was given in a later publication.[4]

PHILIPPUS INGRASSIA

Weber gives in his original work, written in Latin, the following description: "If both ears are closed by the hands, one's own voice is better heard than with open ears. If one ear is closed, one's own voice is better heard by the closed ear. If a tuning fork is pressed against the teeth and the mouth is closed as firmly as possible and the ears are closed by the fingers, the tuning fork is better heard than with open ears. However, when the meatus of one ear is closed and the other one is open, the sound is stronger heard in the closed than in the open one. ("Si altera auris clausa altera aperta est sonum in aure clausa fortiorem quam in aure aperta audimus.") The first part of this description shows that Ernst Heinrich Weber has to be regarded, in addition, as the discoverer of the well-known phenom-

enon of "autophonia." This fact, to my knowledge, has not been mentioned before. It may be added that his name is connected with the well-known Weber's law, stating the geometrical increase of a stimulus to produce an arithmetical increase of sensation. This law, later corroborated by Fechner, is the principle on which the decibel, the intensity unit of auditory stimuli, is based.

Biographical Note. Ernst Heinrich Weber (1795-1878) was Professor of Anatomy and Physiology in Leipzig. He was the oldest one of the three famous Weber brothers. His brother Wilhelm, the well-known physicist, was his co-worker in the previously mentioned "Wellenlehre". The third one, Eduard, was an outstanding physiologist. He discovered, in collaboration with Ernst Heinrich, the buffer action of the vagus on the heart. This work was presented at the meeting of natural scientists in Naples in 1835.

ERNEST HEINRICH WEBER

Ernst Heinrich Weber, although never interested in the practice of medicine, made the fundamental observation that sometimes in deaf people the tuning fork, when placed at the skull, is better heard in the bad ear. This observation, correlated with his physiological experiment, caused him to believe that sometime in the future the tuning fork would possibly be used for the diagnosis of ear diseases. As a matter of fact, it took only a little over a decade until Weber's prediction was fulfilled. It is to the permanent merit of the French Army doctor, Jean Pierre Bonnafont,[5] to have introduced Weber's experiment as a diagnostic method in clinical medicine.

Thus it took 300 years from the discovery of bone conduction before this phenomenon was used for the first time in medical history for differential diagnosis between peripheral and inner ear deafness. This differential diagnosis, as shown in Weber's original experiment, is based on the lateralization of the sound, ipsilateralization (to the diseased side) indicating conduction deafness; contralateralization (to the good side) indicating perception deafness.

It is obvious that this test is of practical use, particularly in instances of unilateral and simple type of deafness but it does not provide any information if the lesion is qualitatively and quantitatively alike in both ears. It is of little value in instances of combined conduction and perception deafness. In these cases the side of lateralization depends mainly on the predominance of one type or the other, and may differ depending on the pitch of the tuning fork used for the test. It can, however, be of great value if in the course of a middle ear suppuration, the "Weber" which was first lateralized to the diseased side suddenly changes to the good side. In this way the advance of the disease into the inner ear may be signalized.

J. P. BONNAFONT

Biographical Note: Jean Pierre Bonnafont (1805-1885) enlisted as a private in the French Army. He was accepted in the Medical Corps in the year 1827. He graduated as a doctor at the University of Montpellier in the year 1824. Then he took part in the war in Algeria for 12 years. Afterwards he started private practice. He contributed numerous valuable articles, particularly on middle ear diseases. Besides the introduction of the Webers' experiment into clinical medicine, one of his greatest merits is the popularization of the myringotomy.

Rinne's Test. While in the "Weber", the things to be compared are the ability of the bad and of the good ear for lateralization of bone conduction, Rinne in his experiment compared the bone conduction with the air conduction of each side separately.

The following is a translation of the pertinent chapter in the original article "Contributions to the Physiology of the Human Ear" by Adolf Rinne,[6] general practitioner of Göttingen. Rinne gives a description of 22 different experiments referring to the ears. This is the first:

"Experiment Number 1. An experiment which can be easily performed shows how much less the conduction by the skull bone

is than the normal conduction by air, ear drum, etc. This is so even when the sounds are produced by a rigid body, immediately attached to the skeleton. I press a vibrating tuning fork against the upper incisors and keep it in this position until the sound, which was in the beginning very clear, becomes inaudible to me. Then I hold the tuning fork in front of the external ear and I again hear the sound with great intensity. Only after a considerable time does it disappear from there. In every person with healthy ears on whom I repeated this experiment the result was the same. If put at the lower jaw, the tuning fork can be heard a little bit longer. This is to be explained by the position of the joint in close vicinity to the ear."

Dr. Rinne then makes an "additional note" (Anmerkung). This reads as follows: "This experiment may be used for diagnostic purposes in cases of deafness. If the result of this experiment performed on a deaf person is the same as on a normal one, then we are justified in concluding that the condition of the entire conduction apparatus is normal. Therefore, the auditory nerve must be the diseased part. If, however, the patient hears the sound transmitted by the bone as long or even longer than he hears it in the normal way, then we have to assume a disease of one of the parts of the conduction apparatus including the membrana of the fenestra ovalis."

H. AD. RINNE

In spite of his exact description and advice on clinical utilization, it took about a quarter of a century until the Rinne test became popular as a clinical method. Undoubtedly some shortcomings in this test diminished its value as a diagnostic procedure. It was to the merit of August Lucae and particularly of Friedrich Bezold to have advanced Rinne's experiment as a useful clinical device. Bezold's systematic and intensive studies particularly were of fundamental importance for the evaluation of this test. He measured the time the vibrating tuning fork is heard by air in comparison with the time it is heard by bone. He called the former *t*, the latter *T*. When

t minus *T* was a figure above zero, the difference was a positive number, designating the Rinne as "positive." In the opposite case, *t* minus *T* being a negative number, the Rinne was called "negative."

It is obvious that even this concept of Bezold is unable to eliminate sources of wrong interpretation lurking in the Rinne test. It becomes clear that in an instance of conduction deafness of slight degree, air conduction (AC) may still be somewhat better than bone conduction (BC). On the other hand, in instances of unilateral nerve deafness, it may be that BC is greater than AC, this being due, of course, to the normal bone conduction on the good side by which sound is transmitted to the diseased side. The differentiation, however, will usually be made easily by using the Weber and the Schwabach tests. The former, of course, will present itself by ipsilateralization in the first case, and contralateralization in the second case. The latter, as I shall indicate later, will reveal an increase of bone conduction in the first case and a decrease or a normal one in the second case.

Our students frequently have a hard time in remembering the meaning of "positive" and "negative" in connection with the Rinne test. They cannot possibly be blamed for this. It was felt by Adam Politzer, the father of modern otology, that this terminology was rather confusing. That was the reason that he suggested at the meeting of the Society of the Otologists of Southern Germany in Munich, May 25, 1885, that the terms "positive Rinne" and "negative Rinne" be replaced by the terms "Rinne present" or "Rinne absent". "For", as he said, "the negative 'Rinne' means a positive result and vice versa." Confusion can, in my opinion, best be avoided by simply recording the results of the Rinne test as AC>BC, or BC>AC, as the case may be. Even that can be shortened by calling the former "AB Rinne", the latter "BA Rinne."

Biographical Note: Rinne* was born in the year 1819, studied in Göttingen and graduated in 1846. He worked first as a general practitioner and was later employed in an asylum for the insane. He died in 1868 of dysentery. A list of some of his papers gives a picture of his unusual scientific mind: "On the Organ of Voice and

*I wish to remind the reader that Rinne spelled his name without an accent on the "e". This accent appears frequently even in textbooks. It was probably caused by a misprint of an accent for an apostrophe in the expression, "Rinne's Test". The name Rinne is to be pronounced with the inflection on the "i", since it is a German and not a French name. This is of some importance because there exists a French otologist, a contemporary of Rinne, with a name sounding somewhat similar. The name of this man is A. René. He wrote "L'audiometre application du telephon à la mesure de l'acuiti auditive".[7]

the Formation of Voice," 1850; "Contributions to the Physiology of the Ear," 1855; "On the Shape of the Firmament," 1864; "Materialism and the Ethical Desire," 1868.

Schwabach's Test: Numerous critical studies of the tests of Weber and Rinne were published after their first presentation. It has been already mentioned that some of them were even rather justified. It was, however, Schwabach who presented one of the most exact and objective studies in this connection, adding a new method of evaluation of bone conduction for differential diagnosis.

In the year 1885 he published the report on his investigations in the classic article, "On the value of Rinne's experiment for the diagnosis of the diseases of the hearing apparatus."[8]

Schwabach examined 104 cases with altogether 164 affected ears, using two different tuning forks. He found the reliability of the "Rinne" and the "Weber" to be considerably restricted, the result of the "Rinne" very much dependent on the tuning fork used. Furthermore, he found that the tuning fork is heard by the conduction deaf ear even when placed at the good side. He, therefore, examined at the vertex. Thus he found the "Rinne" to be negative only in 56.6 per cent of the cases of peripheral deafness. "It seems to me," he wrote, "of considerable value for the differential diagnosis between affections of the conduction and those of the perception apparatus to take into consideration the difference of time, how much longer or shorter a tuning fork may be heard when placed at the vertex of a deaf person as compared with the time it is heard when placed at the vertex of a person who hears normally." Schwabach found this time to be prolonged in 94.4 per cent of cases of conduction deafness, even when a positive "Rinne" was present in 73.6 per cent.

He came to the conclusion that his method offers diagnostically useful results in a greater number of cases than the methods used up to that time. He admitted, however, that none of the three methods is in itself good enough for always answering the question of whether we are dealing in a given case with an involvement of the conduction or the perception apparatus. They are, however, usually significant when used in conjunction with each other.

Biographical Note: Geheimrat Dr. Dagoberth Schwabach (1846-1920) started as a general practitioner in Berlin. In 1872 he began his training in otology as a pupil of Troeltsch and Lucae. Although not connected with the teaching staff of a university, he was held in high esteem by his contemporaries and honored by the presi-

dency of the German Otological Society at the meeting in Dresden, March 14, 1910. At this historical meeting Robert Bárány suggested the fenestration operation for otosclerosis. Schwabach was among the first ones to observe nystagmus as caused by ear diseases (1878). The devising of the test just described made his name immortal.

CONCLUSIONS

In spite of many objections, the Weber, Rinne and Schwabach tests have proved themselves to be the most valuable methods for differential diagnosis between conduction and perception deafness. Even regarding the advances which have been achieved by modern audiometric and other methods of hearing examination, the dictum of Friedrich Bezold still holds good: "If one would reject these tests, it would mean a retrogression from decades of successful research."

The simple phenomenon of bone conduction first observed 400 years ago has turned out to be a discovery of eternal value.

Much of the historical data as weil as the portraits have been obtained from Adam Politzer's "Geschichte der Ohrenheilkunde".[1] In addition, numerous other sources have been used. It is regretted by the author that no portrait of Schwabach could be obtained even after a careful search of the literature.

3507 Burnet Avenue.

REFERENCES

1. Politzer, Adam: Geschichte der Ohrenheilkunde, Stuttgart, 1907.

2. Vesalius, Andreas: De corporis humani fabrica, L. 1, cap. 39.

3. Weber, Ernst H., and Weber, Wilhelm: Wellenlehre auf Experimente Gegrundet oder über die Wellen tropfbarer Flussigkeiten mit Anwendung auf die Schall- und Luftwellen, Leipzig, 1825.

4. Weber, Ernst H.: De pulsu, resorptione, auditu et tactu, Leipzig, 1834.

5. Bonnafont, Jean Pierre: Emploi due diapason dans le traitement des affections de l'organe de l'ouie, Compt. rend d. l'Acad. d. sc., vol. 20, 1845.

6. Rinne, Adolf: Contributions to the Physiology of the Human Ear, Prager Vierteljahrschr. f. d. praktische Heilkunde 1:71-113; 2:45-72, 1855.

7. René, A.: L'audiometre application du telephon à la mesure de l'acuiti auditive, Gaz. d. hop. Par., 1880, p. 644.

8. Schwabach, Dagoberth: On the Value of Rinne's Experiment for the Diagnosis of the Diseases of the Hearing Apparatus, Ztschr. f. Ohrenh. 14:61-148, 1885.

Clinical Notes

XXXVI

CONTACT ULCER OF THE LARYNX

A PERSONAL EXPERIENCE

J. W. JERVEY, M.D.

GREENVILLE, S. C.

Contact ulcer of the larynx is not a common disease. Indeed I have never seen a case to recognize it. In righteous indignation one may at once and with some reason demand the excuse for this paper. The reply is simply this, I have had a contact ulcer of the larynx, and the thought occurred that personal observation as to its incipiency, course, and residual symptoms would be worth reporting.

To begin with it is obvious that the direct cause of the condition is trauma from constant or improper use of the voice. Jackson has emphasized the "hammer and anvil" effect of the two vocal processes hitting each other during phonation. In my case the ulcer came on as the result of a long-continued tendency to clear the throat at frequent intervals. Combined with this was the fairly continuous use of the voice in otolaryngological practice where the patient turnover is rapid and there is a good deal of conversation necessary. Most writers have emphasized the point that nearly all cases occur in inveterate talkers.

Primary etiology may or may not have been an upper respiratory infection, though none was proved. At any rate the direct cause of the ulcer was bruising from often forcible efforts to clear the larynx, the prolonged overuse and abuse of the voice. The lungs and the sinuses were consistently negative clinically and on x-ray examination, and the Wassermann test was negative.

For how many months the affection existed before ulceration occurred it is impossible to say. However, after a longer or a shorter period the first evidence of real trouble was a feeling of fullness in the hypopharynx and a very definite tendency of the larynx to suddenly close in spasm during phonation. This was exceedingly annoy-

ing and passed off on swallowing or clearing the throat. Accompanying the spasm was a sensation of extreme dryness and pain. Later on these spasms became more frequent, often occurring when I was silent, and were associated with a moderate amount of pain on swallowing. The pharynx and the larynx were repeatedly examined at this time but in spite of my insistence that something was wrong there was nothing definite to be observed. The pain on swallowing and the spasm which frequently cut off speech persisted for some weeks without improvement and then the pain definitely localized in the right side, and without suspecting the diagnosis I told my father exactly where the lesion was. I felt as if it were on the inner surface of the right arytenoid cartilage. You may say it could not be localized so accurately by symptoms alone, yet such was the case, and when he looked just there, a small shallow ulceration over the vocal process of the right arytenoid was observed. Dr. V. K. Hart made the diagnosis of contact ulcer of the larynx.

By this time the pain was quite severe on swallowing and definitely radiated to the right ear which was found normal. At all times there was a feeling of fullness and discomfort in the hypopharynx. There was at no time any cough or hoarseness, yet most writers emphasize the latter as a prominent symptom. Occasionally one or two words, either at the beginning or the end of a sentence, could be vocalized only with exceptional effort or after clearing the throat or swallowing. There was no difficulty in respiration at any time.

No local treatment was employed but absolute vocal rest was advised and very conscientiously carried out for three weeks. A pad and pencil were used and an active practice was continued. By the end of the first week the pain was gone both in the throat and the ear, and there was no spasm. Most patients with contact ulcer are not so fortunate. Gradually the use of the voice was increased, but it was found that spasm during phonation reappeared occasionally at what always seemed to be the most inopportune times. Over a period of about six weeks the ulcer healed completely, but the spasm continued occasionally, perhaps three or four times a day and later less often, for several months.

About three years later there was a mild recurrence after a long siege of tracheitis with considerable coughing following an acute upper respiratory infection. This attack cleared up in about ten days with vocal rest and was typical of the first in all its manifestations. Again, most patients are not so fortunate.

To my discredit it must be said that the habit of clearing my throat persists and the more often it is done the greater is the desire to repeat it and the more discomfort there is. The possibility of recurrence is dreaded, the voice tires very easily, and, of course, whenever anything seems to be wrong, reassurance is imperative.

It cannot be said that the throat has ever been perfectly comfortable or as strong as it should be since the first episode. It appears that this feeling of uneasiness and at times even actual discomfort must be accepted along with the possibility of recurrences, especially with vocal abuse. Fortunately, for my own peace of mind the incidence of malignancy in these cases is extremely low, Jackson reporting less than one per cent.

Contact ulcers seem always to occur posteriorly and commonly over the vocal process of the arytenoid cartilage which is often exposed. The condition may be unilateral or bilateral, and healing is usually slow, due to exposure of the cartilage and the repeated trauma of the arytenoids each by the other during the act of phonation.

Complete vocal rest is the best if not the only treatment. Local therapy is of no avail but if granulations form, these may be removed carefully with sharp biting forceps. It is said that these granulations are very friable and easy to remove without pulling or tearing.

For the most complete study on record you are referred to the excellent article by Jackson and Jackson.[2] There are only a few reports in the literature, but it is evident that the condition is much more common than is generally supposed.

Truly it is an ill wind that blows no good and it is my earnest hope that my readers may profit from my experience without the necessity of sharing in it.

101 CHURCH STREET.

REFERENCES

1. Peroni, Achille: Contact Ulcer of the Larynx, Arch. Otolaryng. 17:741-746 (June) 1933.

2. Jackson, C., and Jackson, C. L.: Contact Ulcer of the Larynx, Arch. Otolaryng. 22:1-15 (July) 1935.

3. Woodruff, G. H.: Contact Ulcer of the Larynx, J. A. M. A. 106:1562 (May 2) 1936.

4. Thomas, W. C.: Contact Ulcer of the Larynx, West Virginia M. J., vol. 34, June, 1938.

5. Farrior, J. B.: Contact Ulcer of the Larynx Developing After Intratracheal Anesthesia, Arch. Otolaryng. 36:239 (Aug.) 1942.

XXXVII

UNILATERAL EXOPHTHALMOS

CASE REPORT OF AN UNUSUAL RESULT

JOSEPH A. BUDETTI

CAPTAIN MEDICAL CORPS, ARMY OF THE UNITED STATES

On January 10, 1944, a female civilian dependent reported to the Ear, Nose and Throat Clinic at Oliver General Hospital with a complaint of drooping of the left eyelid, and a somewhat persistent headache in the left periorbital region, plus a feeling of malaise and some lassitude, present since Christmas of 1943. There was no associated nausea or vomiting. Moderate restlessness, but no muscular weakness, was reported. She gave a further history of a recurrent running ear on the right side since the age of 15. The patient was a normally developed white female, 25 years of age, with one child born in December 1942. Her past history was otherwise negative except for a history of measles and of having been a diphtheria carrier in childhood, at which time a tonsillectomy was done.

Examination at that time showed an apparent narrowing of the left palpebral fissure with a relative enophthalmos but no change in the pupil. A slight amount of foul exudate was present in the right ear. The drum showed no injection, but a large perforation was noted in the posterior superior portion, with retraction about the perforation. Hearing on the right was 3/20 for conversational voice, and 0/15 for whispered voice. Several small tonsil remnants were present on each side. The ear, nose and throat examination was otherwise negative.

The eye consultant reported a 3 mm. decrease in the left palpebral fissure, with no weakness of elevation of the upper lid. A slight tenderness on pressure over the left supraorbital ridge was elicited. Exophthalmometer readings were 16 mm. on the right and 13 mm. on the left. The pupils were equal and reacted well to light and accommodation. The media and the fundi were entirely normal. Extra-ocular movements and muscle balance were normal, with no nystagmus seen at that time. No sensory disturbance, such as anhidrosis, was noted by the patient or the examiner on the left side of the face. The neurologist reported the same drooping of the left lid; normal Babinski, abdominal and deep reflexes; no facial palsies;

no disturbance in coordination. Horner's syndrome was ruled out by the neurologist because of the lack of anhidrosis and the normal pupils. Laboratory studies, including complete blood count, differential leukocyte count and sedimentation rate, were all reported negative.

The patient was again seen on January 19 and the symptoms and the findings were unchanged. On February 2, however, the ptosis of the left eye had disappeared. Both upper eyelids were level but there was still a slight difference in the prominence of the eyeballs. A left enophthalmos was still considered present at that time. The patient was reexamined by the Eye and Neurological Departments on February 14 and no change was noted.

Then, on March 2, the patient reported recurrent, daily, afternoon headaches during the previous week. The pains had been sharp and severe and the patient had vomited once during that week. She also reported a feeling of numbness in both lower extremities. It was then felt that the right eye was prominent and that the left eye appeared normal. The relationship had, therefore, changed from enophthalmos on the left to exophthalmos on the right. The exophthalmometer measured the right eye prominence at 16.5 mm., the left eye prominence at 13 mm., with no pathological changes of the fundi, no chorioretinitis, normal fields, and normal tension. Exophthalmometer readings later varied to right 17 mm., left 14 mm.; right 16 mm., left 15 mm.; right 17 mm., left 15 mm.; but no change in the appearance of the eyes was noted on gross examination. The right exophthalmos persisted.

Throughout this time, the right ear had shown a slight persistent discharge in spite of boric acid in alcohol ear drops and sulfathiazole insufflations. A neurological examination in March confirmed the lack of ptosis on the left and the widening of the palpebral fissure on the right. In addition, slight horizontal nystagmoid jerks were reported present on both sides, but the examination was otherwise negative. Reexamination on March 8 gave identical findings. The neurologist considered the chief possibility as aneurysm; the otorhinolaryngologist considered a brain abscess. An operation on the mastoid was recommended by all concerned.

The symptoms of headache, nausea, and weakness of the lower extremities lasted for only one week, and cleared spontaneously.

Re-examinations in May and June, at Oliver General Hospital, showed no change in the condition except for two days of right occipital headache about May 24, with slight temporary blurring

of vision on May 25. The condition cleared spontaneously again. Because of the possibility of an intracranial infection, the patient was referred to Lawson General Hospital for electro-encephalography. She arrived there on July 9, 1944, and the following findings are reported on the final summary from Lawson General Hospital:

"On admission, nerological examination revealed a fine nystagmus with both right and left lateral gaze. The pupils reacted equally. The right eyelid would not close completely and there was noted a slight weakness of the muscles of the right side of the face. All deep reflexes were hyperactive on the right. The electro-encephalogram showed considerable slow activity of the generalized type, but no localization was noted. No bruit was heard about the right orbit. The neurologist stated that no diagnosis could be made. He suggested spinal punctures and arteriograms but these procedures were not performed at that time. Medical consultation to rule out Graves' disease revealed no evidence of hyperthyroidism. The consultant stated that, at that time, the diagnosis was not warranted. Laboratory examinations were reported as follows: Basal Metabolic Rate minus 21%; Urinalysis, Blood Count, and Kahn test were normal; Blood Cholesterol 165; Icteric Index 4.5; Hematocrit 41% volume of packed cell; fragility test normal; blood studies for blood dyscrasia negative. Various x-rays of the skull including special views for the optic foramen and internal auditory meatuses were normal. X-rays of the sinuses were also normal.

"Ear, nose and throat examination confirmed the presence of the large perforation of the right ear drum and the foul discharge. X-ray of the mastoids confirmed the sclerosis of the right mastoid.

"Ophthalmic examination of the fundi, fields, vision, and tension were essentially negative, which confirmed the reports of the eye examination at this hospital. There was noted a difference in the width of the palpebral fissures, which varied from time to time. Readings with the exophthalmometer were R-20 mm. and L-16 mm. at one time; R-15 and L-14 mm. another; and still later R-16 and L-15.

"The neurosurgical consultant felt that the only positive findings were the proptosis and the facial weakness of the right side. He further felt that the facial weakness was due either to the chronic granulation tissue within the right ear or a cholesteatomatous mass which was compressing upon the facial nerve. It was his opinion that adequate surgery should be performed upon the right diseased mastoid with reexamination after three or four months for further

study at that time, either by pneumoencephalogram or by arteriogram of the cerebral vascular circulation, if no improvement were noted."

The patient was then returned to Oliver General Hospital, at her request, for an operation on the right mastoid. A final stereoscopic x-ray check-up of the facial bones about the right orbit was made and the films were reported entirely negative for any possible periorbital tumor. Such laboratory work as was repeated was found to be essentially the same. The sedimentation rate ranged about 12 mm.; the basal metabolic rate was minus 18. Culture of secretions from the ear before operation showed proteus vulgaris and streptococci.

The combined opinions of the medical, surgical, neurological, and ophthalmological and otorhinolaryngological services of both Lawson General Hospital and Oliver General Hospital were as follows:

1. The patient had a chronic otitis media and mastoiditis on the right side. Secondarily, there was possibly some extension or cholesteatomatous erosion which may have been a factor in causing the weakness of the right facial nerve, plus a possible circulatory effect upon the vascular structure behind the right eye.

2. The exophthalmos and the widening of the palpebral fissure varied from time to time, suggesting a possible circulatory phenomenon rather than an orbital or brain tumor. It was considered possible that the weakness of the right facial nerve exaggerated the true exophthalmos and the widening of the palpebral fissure on the right side.

3. Neurosurgical and neurological examinations failed to reveal the presence of any intracranial condition such as a brain tumor or a brain abscess.

4. Eye examination failed to reveal evidence of any pathological changes of the eye or the retrobulbar region; except for the wide palpebral fissure and variable exophthalmos of the right eye, no eye diagnosis was made.

The recommendations of the combined services were that the patient should have: (1) a radical mastoidectomy performed on the right side and special observation made for involvement of the facial canal; (2) re-examination in three to six months with further encephalographic studies if necessary at that time.

On August 14, a right radical mastoidectomy was done and a small beginning cholesteatoma was found in the attic region. No

evidence of erosion of the facial nerve canal or direct pressure on the facial nerve was noted. A culture of secretion taken from the mastoid produced proteus vulgaris and staphylococcus aureus. The mastoid incision healed rapidly but the discharge from the ear persisted. Penicillin packings via the ear canal were used postoperatively.

The patient was discharged from the hospital twelve days after the operation and was then followed at the Ear, Nose and Throat Clinic on an out-patient basis. A series of penicillin packs, tyrothricin packs, sulfathiazole insufflations, and azochloramide packs were used successively for treatment of the persistent ear discharge without benefit. In October 1944 a series of seven x-ray treatments of 75 r. each were started for a total of 525 r. and by the middle of November the ear had become entirely dry. There was a slight recurrence for four days in the middle of December 1944 with rapid spontaneous cessation of the discharge. The patient was again seen on December 28. At that time, the ear was dry and the eye showed no change. However, on January 11, 1945, the patient returned for a check-up and the right eye had receded almost to normal. The exophthalmometer readings were 15 mm. on each side. The ear had remained dry, all symptoms had disappeared, and the patient was highly pleased with the results. There was a short five-day period of ear discharge at the end of February, which cleared promptly, but no recurrence of symptoms and no change of the eye from its normal state. The patient has been seen repeatedly until July 1945 when she moved to Rome, Georgia. Her condition has remained good. She has been examined by the Ear and the Neurological Departments since her recovery and all findings have been entirely negative. Exophthalmometer readings have been repeatedly found to be 15 mm. on each side.

CONCLUSION

A case of unilateral exophthalmos with an unusual result is recorded. The relationship of the right exophthalmos to the chronic right otitis media is problematical but, chronologically, the cure of the eye condition followed immediately on the final curing of the ear condition. It is not believed that the relatively superficial x-ray treatments produced any effect other than definitely contributing to the clearance of the middle ear and mastoid infection.

421 HUGUENOT STREET

NEW ROCHELLE, N. Y.

XXXVIII

NECK INFECTIONS

J. Charles Dickson, M.D.

Houston, Texas

Many articles have been written about neck infections. A great deal has been said about the various fascial planes and possible spaces where the infection might spread. However, when one of these cases has to be treated it is always a serious and difficult problem. This paper is written to give the case histories of three patients, seen about the same time, and to discuss the anatomy involved. I will deviate from the usual method in that I will first give the case histories and then discuss the anatomy.

Case 1.—Mrs. L. C., age 41, was seen August 18, 1944, with a history of having had several left upper teeth extracted three weeks previously. Since then she had developed a swelling of the left side of the face and neck with considerable pain and fever. The dentist had treated her, giving small doses of sulfathiazole and incising the operative area. Examination showed a very toxic patient who was unable to open her mouth more than one-half inch. There was a marked swelling on the left side of her throat and around the left parotid area. She was placed on penicillin, sulfadiazine, x-ray therapy, hot packs and irrigations.

The temperature became septic in character with a drop in the red blood count and an increase in the white blood count. A blood transfusion was given. Five days after admission the condition was much worse and an exploratory operation was done on the left parapharyngeal space. No pus was found, only brawny induration and edema. The same treatment was continued. Twelve days after her admission to the hospital, the patient was still losing ground. The neck was re-opened with negative results. The swelling above the left tonsil was incised and pus obtained. Smears showed staphylococcus, pneumococcus, streptococcus and influenza bacillus. Culture was positive for staphylococcus, pneumococcus and streptococcus. This opening drained for three or four weeks. Seventeen days from

---

From the Department of Otolaryngology, Baylor University College of Medicine.

Read before the Texas Ophthalmological and Otolaryngological Society, San Antonio, Texas, Dec. 14, 1945.

date of entry the patient was still going downhill, became irrational, and developed signs of intracranial involvement. A brain surgeon was called in consultation and he advised an operation. A large brain abscess was found in the temporal lobe and the subdural space.

From this time on, the patient began to improve and made a slow recovery, leaving the hospital on the fiftieth day. The patient was seen two weeks ago. With the exception of scars in the operative areas, no evidence could be seen of the previous trouble. The rest of her teeth had been extracted (by another dentist) and she was wearing a false denture. She was given 4,100,000 units of penicillin and 1240 grains of sulfadiazine.

She had the typical signs of infection in the parapharyngeal area—sepsis, swelling internally and externally, and trismus—yet no pus was found here. The infection probably travelled up from the pterygomandibular region to produce a brain abscess.

CASE 2.—Mrs. M. A., aged 37, came in the office July 19, 1944, with a history of having had teeth in the right upper jaw extracted two weeks previously, and of having developed a swelling over the right side of the face with considerable pain. She was sent to the hospital, and hot packs, sulfadiazine and x-ray therapy were given. In four days the symptoms had improved and the patient was dismissed. The condition was watched for two weeks at the office. Suddenly she became much worse, having had a chill, with a temperature of 106° F. The patient was re-admitted to the hospital and sulfadiazine, penicillin, x-ray therapy, blood transfusions, hot packs and irrigations were given. The pain increased over the parotid area and the mandibular joint on the right. Five days after the second entrance, an abscess developed in the right parotid region and this was drained. Repeated x-ray films of the jaw and maxilla did not show any pathological changes in the bone.

The symptoms improved to the point where the patient was again dismissed on the twenty-fifth hospital day. She continued to visit the office with the abscess wound draining. One week after the second dismissal, all the old symptoms returned and the patient was re-admitted for the third time. An x-ray film now showed considerable involvement of the right mandibular joint. An operation was done exposing the joint and the ramus of the mandible. A large amount of pus was found in this entire area. The joint was completely involved and no periosteum could be felt by finger and probe on the ramus of the mandible. Soon after this operation the patient also developed a discharging right ear, which appeared to come from the canal anteriorally near the drum. The postoperative course

was fairly uneventful and she was dismissed for the third time on the twenty-first postoperative day. She had been given in all 2,220,000 units of penicillin and 1960 grains of sulfadiazine.

Since then the patient has been seen a number of times at the office. She has from time to time a discharge from the fistula in the ear canal. She is able to open her mouth more than an inch. X-ray films show a badly damaged joint but no evidence of osteomyelitis.

This patient evidently had an infection in the pterygomandibular area which spread to the parotid space and drained externally. She too had the typical signs of parapharyngeal space infection—sepsis, trismus, and local swelling, both internally and externally.

CASE 3.—Mrs. L. H., aged 53, was seen August 30, 1944, with a history of having had a left upper molar tooth extracted two weeks before. Swelling and fever had developed in three days and increased steadily since. The dentist had given her small doses of codein and sulfathiazole. In the past history, she stated that she had been a diabetic for many years, had had a cancer of the cervix which was treated with radium, and was operated on for appendicitis and a ventral hernia. There was, on admittance to the hospital, a marked swelling of the left side of the face and neck. The left lateral wall of the pharynx was pushed toward the median line. She could only open her mouth about one-fourth inch. The temperature was 103° F. Treatment consisted of hot packs and irrigations, sulfathiazole, penicillin, x-ray, neoarsphenamine injections, blood transfusions and anti-diabetic measures. Four days after entering the hospital the condition was much worse. An operation was done, opening up the left pharyngeal space where about three ounces of pus were found.

Her general symptoms improved temporarily but the infection continued to spread down her neck and it was necessary as time went on to make other openings. At one time there was drainage from five fistulas. The dressings were saturated with pus in two to three hours and two or three ounces could be expressed from the openings. The diabetes was very hard to control. The patient's condition gradually became worse over the next three weeks. At this time, four weeks after admittance, drainage tubes were placed in the neck and Dakin's treatment started. This seemed to be the turning point and she gradually began to improve. After being in the hospital for 65 days she was discharged with only one small fistula draining. Her general health was good. The discharge stopped soon after her going home. At present the neck is scarred and the skin is retracted and discolored. The muscles are smaller than on the right

side. This patient had 6,500,000 units of penicillin and 2340 grains of sulfathiazole.

The history since this illness has been unusual. Six months later she developed a maniac depressive psychosis. Fifteen shock treatments were given with apparent cure. Three months after this she fell and had a compression fracture of the third lumbar vertebra and was in a cast for a number of weeks. The diabetes now seems to be under control.

This patient had an infection very similar to the infection of the others. The symptoms at first were the same. The spread, however, was down the neck (as usually happens in these cases). The diabetes was a serious handicap. It is of interest to note the number and variety of illnesses this woman has had and from which she recovered.

Comment. The symptoms are varied, depending on the area involved. However, there are four main symptoms common to all cases: trismus, with the neck and head held rigid when turning the body; induration and swelling about the angle of the jaw; sepsis and toxemia; and medial bulging of the lateral pharyngeal wall.

The source of infection may be any tissue or organ in the head and neck—teeth, tissue in which there have been injections for removal of teeth, sinuses, tonsils, pharynx, salivary glands, ears, lymph nodes, tissue in the mouth and throat which has been incised. These three cases were probably caused by infection following the injection of an anesthetic to remove an upper molar tooth.

There are usually numerous organisms involved in the infection. Vincent's organism is frequently present in conditions arising from the teeth.[2] Beck[1] reports the following organisms found in 24 cases: streptococcus, streptococcus veridans, hemolytic streptococcus, staphylococcus, staphylococcus albus, staphylococcus aureus and Vincent's spirochetes. These three cases had staphylococcus, streptococcus, pneumococcus and a gram-negative bacillus.

The complications are many. The condition may spread to the brain or meninges, to other spaces in the neck, down into the chest involving the heart, mediastinum and lungs, and to other parts of the body by metastasis. It may erode a blood vessel and cause death from hemorrhage. The larynx may be involved and the resulting dyspnea require a tracheotomy. The abscess may rupture over the parotid, in the ear canal or in the temporal region. Osteomyelitis

may develop in the nearby bones. The patient may die from sepsis and toxemia. In these cases the infection spread to the meninges and brain in Case 1, to the parotid region and ear canal in Case 2, and to the lower neck by way of the carotid sheath in Case 3.

The main spaces involved are five in number. Actually these spaces are only potential spaces. They dilate when an infection occurs and can become quite large. They are composed of loose areolar tissue and are bounded or limited by dense fibrous tissue or fascia.[4, 7] This fascia covers the nearby muscles, glands, nerves, and blood vessels and is attached to the base of the skull, the mandible, the hyoid bone and the vertebrae. The five spaces are the retropharyngeal, the sublingual and submandibular or submental, (Ludwig's space), the parotid, the lateral pharyngeal, parapharyngeal or pharyngomaxillary, the downward extension of the parapharyngeal space around the carotid sheath into the neck and mediastinum.

The retropharyngeal space lies posterior to the pharynx between the fascia of the pharynx, and the prevertebral fascia. Iglauer[4, 5] and Weintraub[7] describe this space and state that the infection is caused by the suppuration of lymph glands. Another cause is an injury to the posterior pharyngeal wall. Also infection in nearby areas may drain into this space. If not drained, the infection travels down the neck in front of the vertebrae into the mediastinum.

Ludwig's angina is now generally considered as an infection in the sublingual and submaxillary space either above or below the mylohyoid muscle. This space communicates with the parapharyngeal space near the submaxillary gland back of the mylohyoid muscle.

The parotid space is around the parotid gland. It connects with the parapharyngeal space beneath the parotid gland near the angle of the jaw.

The pharyngeal space is a triangular space with the base against the skull and the apex at the greater cornu of the hyoid bone.[3, 6] The boundaries are: internally, the lateral pharyngeal wall; externally, the ramus of the mandible and a portion of the capsule of the parotid gland; anteriorly, the internal pterygoid muscle, and posteriorly, the prevertebral muscles and fascia. This space contains the styloid process and its muscles, the carotid sheath, several cranial nerves and numerous lymph nodes. Some anatomists have divided this space into an anterior part, anterior to the styloid process, and a posterior part, posterior to the process. At the apex this space communicates with the sublingual or submandibular space anteriorly, with the

parotid space externally and with the space along the carotid sheath inferiorly. Near this space lie the pharynx, tonsils, and the pterygomaxillary region.

The carotid sheath space extends from the parapharyngeal space above down into the chest below.

Infection may travel from space to space, thus involving the entire neck. The surgical approach to the various spaces depends on the space involved.

The retropharyngeal space is usually drained through the mouth. The sublingual space is reached through incisions under the tongue. The submandibular space is drained by an opening under the chin. The parotid space can be opened by incisions over the gland.

The parapharyngeal space is the most difficult to drain. Mosher[6] and Hall[3] described an excellent approach. These two papers have a number of illustrations describing in detail the anatomy and surgery. Everyone attempting to do this kind of surgery should read the above articles. The space is opened by cutting down over the lower edge of the submaxillary gland, retracting the gland and by blunt dissection going into the apex of the space. This approach also drains the point where the sublingual, submandibular and parotid spaces open into the parapharyngeal space. After entering the parapharyngeal space one can palpate the styloid process posteriorly. Also from this point the parotid and submandibular spaces can be entered. If the infection is in the front part of the anterior section of the parapharyngeal space it may be drained through an incision anterior to the tonsil near the lower third molar tooth. The posterior section at times in mild cases can be opened from the inside back of the tonsil.

If the infection goes down the carotid sheath space, incisions have to be made over the course of the sheath. This space has been called the "Lincoln Highway" of the neck because it carries infections from above into the mediastinum.

The mortality before the sulfonamides and penicillin appeared was from 40 per cent to 60 per cent.[2] Though these drugs have not eliminated surgery they have definitely increased the percentage of cases that recover.

SUMMARY

1. Three cases of infection in the neck are presented.

2. A brief discussion is given on symptoms, source of infection, causative organisms, complications and medical treatment.

3. A combination of chemotherapy and surgery offers the best hope of recovery. Surgery, when undertaken, must be more than a stab wound. The spaces must be opened, explored widely and good drainage established.

1617 Medical Arts Building.

REFERENCES

1. Beck, A. L.: A Study of Twenty-four Cases of Neck Infection, Annals of Otology, Rhinology and Laryngology 42:741, 1933.

2. Gaines, T. R., and Hatcher, M. B.: Penicillin in the Treatment of Patients with Deep Infections of the Neck, Arch. Otolaryng. 42:1 (July) 1945.

3. Hall, Colby: The Parapharyngeal Space: An Anatomical and Clinical Study, Annals of Otology, Rhinology and Laryngology 43:793, 1934.

4. Iglauer, Samuel: Acute Suppuration of the Spaces of the Neck, J. Michigan Med. Soc. 41:936 (Nov.) 1942.

5. Iglauer, Samuel: Anatomic Pathological Studies of Retropharyngeal Abscess, Arch. Otolaryng. 33:31 (Jan.) 1941.

6. Mosher, H. P.: The Submaxillary Fossa Approach to Deep Pus in the Neck, Trans. Amer. Acad. Ophth. and Otolaryng. 34:19, 1929.

7. Weintraub, J. D.: A New Anatomic and Functional Systematization of the Connective Tissues of the Neck, Arch. Otolaryng. 33:1 (Jan.) 1941.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, December 3, 1945

THE PRESIDENT, DR. JOHN F. DELPH IN THE CHAIR

Sulfonamide Toxicity: Interpretation of Laboratory Findings.

I. DAVIDSOHN, M.D.

(Abstract)

Laboratory tests properly interpreted may help to avoid serious complications in the course of sulfonamide therapy:

The finding of sulfonamide crystals in the urine is of little consequence.

Microscopic hematuria should focus attention on the adequacy of fluid intake and output, and on possible other impending complications, but does not of itself require interruption of therapy.

In the presence of gross hematuria, sulfonamide therapy should be stopped immediately.

The chemical reaction of the urine should be watched in patients receiving alkalies.

Blood counts and differential studies should be made at reasonable intervals.

A blood count prior to initiation of therapy may help to evaluate subsequent findings.

Leukopenia is not a contraindication to sulfonamide therapy, neither is a leukopenia down to about 3,500 white blood cells, when it develops in the course of treatment. Severe granulopenia is an absolute indication for immediate cessation of treatment with the compound used. Another sulfonamide compound may be tried with caution. Acute hemolytic anemia demands immediate cessation of sulfonamide therapy. Equally serious is thrombocytopenia.

Recent reports have shown that acute periarteritis nodosa may occur in the course of sulfonamide therapy. This complication is more common if a horse immune serum is administered simultaneously, especially if serum disease develops.

DISCUSSION

DR. OTTO SAPHIR: In glancing at the tables Dr. Davidsohn has presented, one is struck by the multitude of findings seen at postmortem examination and during clinical and laboratory examination in patients who have received the various sulfonamides. Undoubtedly such a table should make the clinician afraid to use sulfonamides.

What may we find in patients who come to postmortem examination, dying as the result of sulfonamide treatment? There are three factors which should be taken into consideration. First, there are patients who have an idiosyncrasy to sulfonamides in general. Such patients may show reactions during the first treatment and after the first doses of sulfonamide medication. Others gradually become sensitized to the sulfonamides. This we will see more and more because, as you know, during the last few years, after any operation, the field has been sprinkled with sulfadiazine or other of the sulfonamide compounds. What do we find in such cases? Principally, what is called hyperergic inflammation, particularly accentuated in the regions of the joints. We might find this in the region of the blood vessels in the form of a typical periarteritis nodosa. The patient may die as the result of diffuse periarteritis nodosa, particularly if the disease involves the myocardium.

The second factor to be considered is poisoning with sulfonamide drugs, seen in patients who die after having received large amounts. In such patients we see various changes in the hematopoietic system, as Dr. Davidsohn mentioned, and severe changes in the liver in the form of acute hepatitis, or rather a toxic necrosis. We see severe damage in the kidney with blockage of the tubules by the sulfonamide which can actually be demonstrated under the microscope. Hemorrhages in the pelves are common.

As an illustration of the third factor, I would like to mention three types of cases. A patient who had an upper respiratory infection had routinely received sulfonamides. He developed what was clinically called acute rheumatic fever, with increased temperature, increase in sedimentation rate, joint pains and swelling of the joints. He died of bronchopneumonia. At autopsy no evidence of rheu-

matic fever was found; all the symptoms thought to be the result of rheumatic fever were the result of sulfonamide medication.

A second patient also received sulfonamides for an upper respiratory infection. He developed what was diagnosed clinically as acute hemorrhagic glomerulonephritis. Red blood corpuscles and casts were found in the urine; the blood nonprotein nitrogen gradually became elevated and he died from what was thought to be acute hemorrhagic nephritis. Autopsy disclosed that the patient did not have an acute hemorrhagic nephritis, but died of sulfonamide poisoning. It is sometimes very difficult to differentiate between acute hemorrhagic glomerulonephritis and kidney changes due to poisoning with sulfonamide compounds. These two cases show that the sulfonamides may mask the clinical symptoms and lead to an erroneous diagnosis. Such cases are becoming more and more frequent.

There are other cases, particularly in children who develop an upper respiratory infection. The physician may prescribe some sulfonamide medication by telephone. The child is all right until he develops an earache. The specialist is now consulted and makes a diagnosis of otitis media and says, "before doing a paracentesis, let us wait and see what the sulfonamide will do." The otitis media seemingly clears up miraculously and the paracentesis is not done. Although the otitis media has apparently cleared, the child suddenly develops symptoms of acute meningitis or brain abscess. I have seen such cases, and I believe that if sulfonamides had not been used the otitis media would have been treated by paracentesis and the complication would not have occurred. It is true that we see many less operations performed for mastoiditis than before the era of sulfonamide medication, but we still see the fatal complication of abscess in the brain and meningitis, perhaps more so than before the advent of treatment with these drugs.

The sulfonamides should not be used indiscriminately. They should be prescribed only when other therapy fails. Of course, I speak from the point of view of a pathologist who is never asked to prescribe for an acute infectious disease.

Dr. Thomas C. Galloway: Masking of symptoms with sulfonamides should not cloud the diagnosis if we make allowance for the effects of the drug. Because of sulfonamides pediatricians have fewer and fewer ears operated on. I think there can be much impairment of hearing resulting from organization of exudate left after the infection has subsided. I have seen several such cases, and Dr. Wishard mentioned it last year at the meeting of the Triological

Society. Where a child has a frank suppurative otitis media, opening and drainage are necessary to prevent this and other possible complications.

I should like to ask whether sulfonamide fastness is due to inadequate dosage or to sulfonamides given previously.

DR. WALTER THEOBALD: I doubt if we, as otologists, are as great offenders in the indiscriminate prescribing of sulfonamides as the family medical man who is called upon to make a diagnosis and prescribe treatment, often by telephone. Some sulfonamide preparation is on the bathroom shelf, and anyone in the household who has elevation of temperature resorts to the bottle; they are used as freely as salicylates. I have observed patients with recurrent colds, recurrent infections, and have wondered why they were so nonresistant to the common cold. Many times when such a case is studied hematologically we find a low leukocyte count and marked hemolytic anemia.

There is another group on which I would like to have Dr. Davidsohn's comment; these are patients who have been under sulfonamide therapy for some time and do not get over the effect; they have malaise, are below par, tired, fatigued—not a few days but for weeks. Would this present pathologic evidence to you?

DR. SAMUEL SALINGER: Dr. Saphir mentioned the delayed or masked effect of sulfonamides, which brings to mind two clinical items I have encountered which I think worthy of consideration. The first has to do with nausea and vomiting following an operation performed under local anesthesia where sulfonamides have been given. It is difficult to determine whether this reaction is due to the drug or is the result of other factors such as the withholding of fluids for 12 hours prior to operation, plus the effect of morphine and barbiturates usually administered in these cases. Our nasal cases are given sulfonamides for 48 hours following operation. It has been my observation that at least 25 per cent of these patients have marked nausea and vomiting which we have routinely attributed to the sulfonamides. It has occurred to me recently that it might be due to withdrawal of fluids as well as to the reaction from the barbiturates and sulfonamides. It is true that in most cases nausea and vomiting cease when the sulfonamides are withdrawn, yet in some cases nausea has persisted.

The second observation should be of interest to men doing rhinoplasty. Before sulfonamides were given routinely we had a certain percentage of infection—close to 10 per cent of all cases.

Since we have given sulfadiazine for 48 hours following operation we find the percentage of infection greatly reduced, but we are encountering a few cases of delayed infection. Patients used to develop infection within 48 hours; now we send them home in good condition only to find that a certain number develop delayed infection appearing a week or ten days later. This would seem to indicate that the sulfonamides in some cases merely retard the activity of the bacteria which come to life after withdrawal of the drug. It has been my observation that these cases of delayed infection are more serious than those which develop early because they result in more fibrosis and thickening. One is forced to conclude that either the drug should not be given at all, or else its administration must be continued for a longer period of time which, of course, means taking additional risks from the drug itself.

DR. I. DAVIDSOHN (closing): I limited myself to the interpretation of laboratory findings, and did not mean to warn you against the use of sulfonamides. I am trying to help you draw conclusions from what is found in the laboratory so that you may use the laboratory findings as a means of forestalling some ill effects. It is one thing to order laboratory tests and another to interpret them properly; they are of no advantage if not properly interpreted. I have read reports of serious complications following the use of sulfonamides; some of the cases were thoroughly worked up with all possible tests, but in many the proper conclusions were not drawn. For that reason I say correct interpretation is fully as important as correct performance of laboratory tests.

Regarding periarteritis nodosa following sulfonamide therapy, Rich showed that the drug is just one factor. He was able to produce periarteritis nodosa in rabbits treated by horse serum without sulfonamides. Hence, the combined use of horse serum or horse immune serum with sulfonamides is more dangerous than are the sulfonamides alone.

I should like to emphasize what Dr. Saphir and Dr. Theobald mentioned about the danger of sensitizing the patient. A small amount of the drug may be sufficient for this, and the patient may be handicapped in the future when sulfonamides are urgently needed. Some people develop sulfonamide fastness or, more correctly, bacteria responsible for the infection become resistant to sulfonamides. In certain bacterial strains this is possible, and this, of course, proves a handicap to the patient. However, even with due consideration of the complications it is a small price to pay for all the benefits derived

from the use of the sulfonamides. Rational use of laboratory findings will decrease the occurrence and the severity of complications.

Concerning the patient mentioned by Dr. Theobald who developed symptoms of tiredness, fatigue and lack of pep. A drug that depresses the bone marrow and produces changes in various tissues, as mentioned in my paper, is likely to keep the patient below par for a long time, especially if he has been previously sensitized.

In answer to Dr. Salinger, I believe that nausea and vomiting following sulfonamide therapy should be attributed to the toxic reaction of the drug.

What is the explanation of the late infections following rhinoplasty in patients receiving sulfonamides? Let us consider how these drugs act. There have been various hypotheses. According to one, the resistance of the patient is stimulated; this idea has been shown to be incorrect. The present concept is that the growth of bacteria is inhibited temporarily so that they do not multiply. Thus, if sulfonamide therapy is stopped too soon the bacteria again begin to develop, sometimes with much greater virulence than before, because the body did not have an opportunity to develop immunity responses in the absence of the stimulus of the infection. That explains why in some patients insufficient treatment may be injurious and may be followed by a more virulent infection than at the onset.

The External Ear and Drum Membrane in Otosclerosis

FRANK WOJNIAK, M.D.

(This paper appears in full on page 406.)

DISCUSSION

DR. HANS BRUNNER: I think this is an excellent and timely paper. I agree that there is actually no finding of the drum or the external canal which would permit us to make or to exclude definitely the diagnosis of otosclerosis. I might add that microscopic studies of these parts of the temporal bone in cases of otosclerosis furnish the same result. I was, however, surprised to learn that impacted cerumen occurs in patients with otosclerosis about as frequently as in normal individuals. I have always had the impression that impacted cerumen was a comparatively infrequent finding in otosclerosis, but apparently my idea was not correct.

In regard to Schwartze's sign, Dr. Wojniak may be interested in the findings of the late Dr. Schnierer (Monatschr. f. Ohrenh., 1924), who also noted this symptom not only in otosclerosis but also in normals and in other types of deafness, particularly in congenital syphilis. Dr. Schnierer also investigated Schwartze's symptoms after injecting adrenalin through the eustachian tube into the tympanic cavity. He found that the red color usually persisted under these circumstances. Exceptionally, the red color disappeared; this, however, occurred in a person with normal hearing, not in one with otosclerosis. This finding would indicate that the blood vessels which cause the red color of the promontory do not respond to adrenalin. They cannot, therefore, be the common blood vessels of the tympanic mucosa; they must be abnormal blood vessels. Dr. Schnierer presents microscopic findings of a case of this type. The specimens were taken from a man, aged 23, who was observed by Professor Alexander. He had an advanced otosclerosis and a typical Schwartze's symptom. His five brothers and sisters were deaf mutes. After the injection of Schleich's solution into the nasal septum the patient suddenly expired. Autopsy revealed a status thyicmolymphaticus and nephritis.

In this case there was an advanced otosclerosis which occupied the entire lateral wall of the internal ear. The otosclerotic focus was characterized by two findings: It extended to the mucosa of the tympanic cavity, the blood vessels of which were markedly dilated; the otosclerotic focus contained very large blood vessels indicating definite activity of the disease. In this case Schwartze's symptom indicated not only an otosclerosis per se, but an active otosclerosis which approached the tympanic mucosa and caused dilatation of the blood vessels of the mucosa. Although the findings in one case do not allow general conclusions to be drawn, these findings do explain three clinical observations concerning Schwartze's symptom:

1. Schwartze's symptom does not appear in all cases of otosclerosis; it appears only in instances of active otosclerosis.

2. Schwartze's symptom may be present in a case of otosclerosis but may disappear when the activity of the disease subsides.

3. Schwartze's symptom does not respond to adrenalin, because the newly formed blood vessels of the otosclerotic focus do not contain vasomotor nerves.

DR. FRANK WOJNIAK (closing): We found impacted cerumen in two cases of definitely proven otosclerosis.

Abstracts of Current Articles

EAR

Deafness with Undeveloped Mastoid and Tympanic Membrane.

Johnson, Fordyce: Arch. Otolaryng. 42:174-177 (Sept.) 1945.

The author found a group of patients in the Army personnel who complained of deafness of varying degrees which had been present since childhood, but who gave no history of otitis media and in whom the tympanic membrane appeared normal. They had previously been given diagnoses of defective hearing of undetermined etiology. Roentgenograms of the mastoids revealed underdeveloped or undeveloped mastoids. It was concluded that these cases of deafness resulted from infection of the middle ear and mastoid in infancy or early childhood.

HILDING.

Aero-Otitis Media and Loss of Auditory Acuity in Submarine Escape Training.

Shilling, C. W.: Arch. Otolaryng. 42:169-175 (Sept.) 1945.

The author studied the effects on hearing of submarine escape training. He found that 30 per cent of the men taking this training developed aero-otitis media. Each candidate in this school was subjected to about 3.4 atmospheres (50 lbs.) of pressure. This type of trauma leads to acute auditory damage which may be permanent if it is repeated often. All of those subjects showing severe damage were found to have flattening of the eustachian orifices due to lymphoid hyperplasia. If these are eliminated by preliminary nasopharyngoscopy, auditory damage can be reduced to two to three per cent.

HILDING.

Effects of Gun Blasts On Hearing.

Machle, Willard: Arch. Otolaryng. 42:164-168 (Sept.) 1945.

This is a study of the effects of gun blasts from 37 and 75 mm. guns on 45 gunnery instructors. The author finds that the effect on the hearing is similar to that of sustained high noise levels. There is great individual variability in the response to controlled exposure

to gun blast but acoustic injury is common. Potentially serious partial deafness occurs in significant numbers of military personnel. With repeated exposure acoustic damage is cumulative and if the exposure extends over months, recovery to pre-exposure levels of acuity becomes doubtful.

HILDING.

NOSE

Frontal Sinusitis.

Brown, J. M.: Laryngoscope 56:116-119 (Mar.) 1946.

The early treatment of acute frontal sinusitis at the Los Angeles County General Hospital is nasal shrinkage and when indicated infraction of the middle turbinate. Penicillin is started at once and maintained throughout the attack.

If adequate drainage is not established and the symptoms persist, a small opening for the release of pus is made in the sinus floor at its inner angle. A rubber tube is inserted and held by a skin suture. No further local manipulation is instituted for several days; then the cavity is irrigated. If the solution passes freely into the nasal cavity, the tube is removed and the opening closed with one or two skin sutures. If, however, the irrigating solution fails to reach the nasal cavity, an attempt is made to pass a cannula into the sinus through the ostium. If this fails, intranasal procedures are adopted to improve drainage space. Obstructing cells are removed and septal resection may be required before proper drainage space is attained.

The author calls attention to the various complications associated with frontal sinusitis. Subperiosteal abscesses through the floor and the anterior wall are drained at the time of trephination or whenever they develop. Osteomyelitis is usually prevented by early drainage but extension through the posterior wall is encountered regularly.

In the presence of meningeal symptoms, such as persistent severe headache and pain over the frontal area with increased cell count and pressure in the spinal fluid, the posterior wall of the sinus is removed and this permits immediate drainage of intracranial abscess.

A conservative external procedure for management of chronic frontal sinusitis is described. Its adoption presupposes a failure of all conservative intranasal procedures and the elimination of all possible contributing factors of an allergic or endocrine nature. In Brown's technic, the sinus is cleaned out except in the area of the

ostium which is left entirely alone and the good results attained may undoubtedly be attributed to the preservation of the mucosa at this point.

VANALYEA.

The Closure of Oromaxillary Fistulae. A Preliminary Report.

Proctor, Bruce: Laryngoscope 56:46-47 (Feb.) 1946.

The author describes a new method for closure of persisting oro-antral fistulae arising from tooth extraction.

A cone-shaped piece of preserved cartilage is wedged into the tooth socket which is properly prepared by thorough curettement.

The protruding portion of the graft is cut off level with the alveolar process and according to Proctor heals into place without the need of mucosal covering.

The antral infection is managed later by whatever means is required to eliminate the disease.

The results obtained by the author on a limited number of cases have been encouraging. The technic is extremely simple and gives promise of eliminating other more extensive procedures which at the same time have not always proved satisfactory.

VANALYEA.

BRONCHI

Penicillin in the Treatment of Bronchiectasis.

Bobrowitz, I. D., Edlin, J. S., Bassin, S., and Woolley, J. S.: New England J. Med., vol. 234, No. 5 (Jan. 31) 1946.

In a study of 12 cases, penicillin was administered parenterally intratracheally and by inhalation and combinations of these. The concentration in the sputum was highest with intratracheal injection, less with inhalation and lowest with intramuscular injection. Considerable improvement was noted, especially with intratracheal administration, but this did not persist after treatment was stopped. This form of therapy is suggested as a pre-operative preparation for lung resection.

HILL.

Survey of Bronchial Asthma in Soldiers: Bronchoscopic Findings and Incidence of Respiratory Infection.

Zoss, Albert, Neidlinger, William J., and Read, Hilton S.: *J. Allergy*, March, 1946.

The authors of this paper bring out some rather important facts after the observation of 250 cases of perennial bronchial asthma. One very worthwhile observation is that bronchoscopy proved to be of considerable value in deciding the disposition of a case, because if by such an examination it could be determined that chronic bronchial infection existed it was realized that the asthma could not be satisfactorily controlled, regardless of how favorable the case appeared otherwise.

It was also found that 50 per cent of these cases of bronchial asthma showed chronic suppurative sinusitis. Bronchoscopy showed bronchial abnormalities in 89.6 per cent of the cases studied; 49.2 per cent of these were chronic suppurative bronchitis, 38.8 per cent were chronic nonsuppurative bronchitis, and only 1.6 per cent were allergic bronchitis.

McLaurin.

MISCELLANEOUS

Treatment of Allergic Conditions with Pyribenzamine and Benadryl.

Epstein, Stephan: *Wisconsin M. J.* 45:489-496 (May) 1946.

Pyribenzamine and benadryl are two anti-histamine drugs which have appeared in recent years. Their use is based upon the idea that the specific allergic reactions are caused by the liberation of histamine or a histamine-like substance which causes an inflammation of the cells of the conjunctiva and the nasal mucosa, the so-called "shock" organs of the body.

The allergic chain reaction is pictured somewhat as follows:

1. The individual becomes sensitized to an antigen.
2. The sensitized individual produces specific antibodies to this antigen which settle in the cells of the mucosa of the nose and conjunctivae or in the cells of other "shock" organs.
3. Upon renewed exposure to the antigen an antigen-antibody reaction occurs in the shock organ.
4. This antigen-antibody reaction leads to the liberation of histamine or a histamine-like substance.

5. The liberation of this histamine or histamine-like substance produces an inflammatory reaction in the cells of the "shock" organ giving rise to the clinical symptoms.

The effort to break this chain at some point before the liberation of the histamine leads to the use of anti-histamine substances. The first such substance was the enzyme "histaminase" marketed under the trade name Torantil. Its use was successful except in certain food allergies.

More recently two derivations of amina-ethanol and ethylenediamine called pyribenzamine and benadryl have been found to be much more effective in the inhalant type of antigens. These drugs show a very low toxicity when given orally but are quite toxic when given intravenously. Drawing on the literature and also on his own cases Epstein finds that these two drugs are very helpful in controlling the symptoms of hay fever, up to 85 per cent of the cases, but are not so beneficial in the cases of allergic rhinitis due to dust or foods.

The recommended oral dosage is 50 mg. three or four times daily. If the symptoms are controlled, the dose is reduced to one-half or less; if not, it may be doubled.

GROVE.

The Volumetric Incidence of Atmospheric Allergens. IV. A Proposed Standard Method of Gravity Sampling, Counting, and Volumetric Interpolation of Results.

Durham, Oren C.: J. Allergy, March, 1946.

Durham has now presented four articles in the Journal of Allergy, explaining the importance of very accurate measures to determine the volumetric incidence of atmospheric allergens. It seems that the methods for estimating the volumetric incidence of these allergens was quite inaccurate and that the errors were due to many physical factors that had to be considered in devising the proper type of air sampling apparatus.

The device that he has finally found most satisfactory is one that is made of two parallel planes of polished stainless steel with a slide holder raised about one inch above the center of the lower plane. The collecting slide is placed on this holder and exposed for a certain length of time. "The basic index of atmospheric contamination in a given community should depend on aerobiologic data secured from air samples taken on the unobstructed top of a tall downtown building." When pollens are collected by this plan, the

author feels that the volumetric incidence is reasonably accurate and certainly much more dependable than estimates made in the past where there was no standardized method used.

"A method of volumetric conversion of pollen and fungus spore counts is submitted with definite factors for about 40 pollens and one fungus spore. Two sets of conversion factors are listed so that counting may be done on the basis of 1 sq. cm. of slide area or on 3.6 sq. centimeters."

The author has received cooperation in determining these pollen and fungus counts by a number of prominent allergists in this country and it is probable that his plan and apparatus will be adopted rather generally. The importance of all this work is to determine more accurately why patients known to be allergic to certain pollens would react at certain times. It also makes it possible for these patients, if they desire, to seek areas for their vacations or to establish residences where they may be reasonably sure that they would not be disturbed by allergic reactions.

McLAURIN.

Penicillin Aerosalization in the Treatment of Serious Respiratory Infections.

Segal, M. S., and Ryder, C MacI.: New England J. Med., vol. 233, No. 25 (Dec. 20) 1945.

Treatment was ineffectual in six patients with infective bronchial asthma. Clinical cures were obtained in five patients with pneumococcal pneumonia and in one with acute pulmonary infection. Aerosalization was markedly beneficial in five patients with bronchiectasis and this form of therapy was considered more helpful than sulfonamides or penicillin administered parenterally. Of the four patients with lung abscess so treated, one died after surgery, one recovered after surgery, while two recovered without surgery. The authors conclude that penicillin aerosalization is of definite value in the treatment of patients with severe respiratory disease.

HILL.

The Penetration of Penicillin Through Normal and Inflamed Meninges.

Kinsman, J. M., and D'Alonzo, C. A.: New England J. Med., vol. 234, No. 14 (April 4) 1946.

The concentration of penicillin in the spinal fluid was studied in cases with and without meningeal involvement, which had been treated intramuscularly, intrathecally and by a combination of both

methods. While there was some evidence that penicillin in small amounts might penetrate inflamed meninges, this was too inconstant and too erratic to justify intrathecal treatment alone in purulent meningitis.

HILL.

Roentgen Visualization of the Fractured Temporal Styloid Process.

Sinberg, S. E., and Burman, M. S.: Radiology 45:599-602 (Dec.) 1945.

Fracture of the temporal styloid process may occur in diffuse head and neck injuries or in the operative removal of the palatine tonsils. It may be suspected when the patient has difficulty in swallowing or experiences pain between the mastoid process and the mandibular condyle. A method of roentgen visualization is described and resultant radiographs shown. The entire styloid process is clearly visualized by this technique.

JORSTAD.

Cerebral Abscess of Otitic Origin.

Velasco, R.: Rev. de Otorrinolaring. 5:236 (Dec.) 1945.

The author presents a study of nine cases of cerebral abscess arising as a complication of chronic otitis media. In eight cases the abscesses developed by direct continuity, while in one case the abscess is presumed to have been carried by the vascular route. There were no cases secondary to thrombosis of a lateral sinus. The most frequently cultured organisms were streptococcus, staphylococcus and pneumococcus.

The symptoms of the earliest stages are those of encephalitis which continue for approximately one week and are characterized chiefly by headache. Subsequently the symptoms are altered as the abscess becomes encapsulated during a period of about one month. Symptoms following encapsulation are nausea, vomiting and slow pulse. Papillary edema is a late finding and there is no relation between the size of the abscess and the amount of the edema.

The author performs repeated lumbar punctures previous to drainage and does not fear that such procedure may induce rupture of the abscess. The spinal cell count is important, but not of great value in determining the prognosis.

Surgical drainage is the only treatment worthy of consideration. During exploration he relies on the prominence of the dura and the induration to determine the precise area for drainage and searches

freely in all directions for the abscess. His approach to the abscess is through the mastoid following the path of the infection. When the abscess is reached and emptied, drainage is continued by insertion of a Nelaton catheter.

Continued postoperative fever is a sign of additional progress of the infection or retention of pus in the abscess cavity.

If the condition of the patient is such that surgery is demanded before encapsulation is completed, it is done but he advises that a neurosurgeon be called in consultation.

HIGBEE.

Abscess of the Cerebellum.

Otte, J.: Revis. de Otorrinolaring. 5:192 (Dec.) 1945.

The author presents a study of eight cases of cerebellar abscess. He lists three prime considerations which must be borne in mind in the diagnosis and treatment of this disease. These are the type of otitic disease, the symptoms presented and the treatment which naturally is influenced by the other two factors.

In reference to the type of lesion he mentions the frequent association of cerebellar abscess with thrombosis of the lateral sinus. The most frequent pathway by which infection reaches the cerebellum is through the labyrinth with production of labyrinthitis. This was the route of the infection in four of the eight cases reported. In cases of chronic otitis media a complication such as thrombophlebitis or labyrinthitis indicates repeated neurological examinations, observations of the ocular fundi, spinal punctures and examinations of the psychic state of the patient.

Diagnosis of cerebellar abscess is not difficult when it exists as the only complication of chronic otitis media, but as most frequently found it is associated with labyrinthitis, meningitis or thrombophlebitis. Seven of the eight cases reported were complicated by one or more of these conditions.

The classical symptoms of cerebellar abscess are spontaneous nystagmus, atony, asynergia, dysmetria, adiadokocinesis, vertigo and stance with feet far apart. In addition the symptoms which support this diagnosis are occipital pain, slow pulse, emesis, lethargy, loss of weight and somnolence.

Frequently a cerebellar abscess is initiated by chills and high fever which simulate thrombophlebitis. In such instances an important point in differentiating these conditions is that the general appearance of the patient is excellent in thrombophlebitis, whereas

in cerebellar abscess the patient is manifestly sick. A decrease in the leucocyte count in cerebellar abscess signifies encapsulation and must not be considered as evidence of convalescence.

In the treatment of cerebellar abscess the author favors surgical approach through the mastoid and lists his reasons as follows:

1. After performing a simple mastoid operation there is direct entrance into the cerebellum.

2. Artificially produced adhesions between the meninges and the cerebellum are not necessary in this approach. These adhesions have already been created by the inflammatory reaction previous to exploration.

3. Anatomically the cerebellar abscess is most superficial at the level of the mastoid approach.

Whereas formerly there existed great fear of producing new avenues of infection by exploration across the infected mastoid, the use of penicillin and sulfonamide drugs have made this a safe area for drainage of a cerebellar abscess.

HIGBEE.

Thrombophlebitis of the Lateral Sinus.

Otte, Jorge: Rev. de Otorrinolaring. 5:173 (Dec.) 1945.

The author lists the complications of suppurative otitis media in their order of frequency as follows: thrombophlebitis of the lateral sinus, leptomeningitis, encephalitis, intracranial abscess.

In his experience, contrary to the views of many authors, mere exposure of the sinus during mastoidectomy does not induce inflammation within the sinus.

The diagnostic criteria of thrombophlebitis are daily excursions of temperature, chills, no mental abnormalities, no labyrinthine symptoms, no headache and a generally excellent appearance which is very deceptive. Additional symptoms related to the nervous system should suggest intracranial disease particularly cerebellar abscess.

In the surgical care of those patients whose illnesses are without other complications he discusses the question of ligating the internal jugular vein. He favors this technique, but believes that the indications for it will be much reduced by the use of penicillin. This drug he has found much more effective than the sulfonamides. Two

cases of thrombophlebitis are cited in which the sulfonamide drugs were ineffective.

The most frequent complication of thrombophlebitis is brain abscess. The abscess is more apt to localize in the cerebellum if the left lateral sinus has been thrombosed and in the cerebrum in cases of thrombosis of the right lateral sinus.

In general the author favors removal of all diseased tissue, a wide incision of the sinus, adequate dosage of penicillin and ligation of the jugular vein.

HIGBEE.

The Repair of Cranial Defects with Special Reference to the Use of Cancellous Bone.

Carmody, J. T. B.: New England J. Med., vol. 234, No. 12 (March 21) 1946.

When the defect is not too large the author feels that autogenous cancellous bone is the material of choice for closing skull defects. While tantalum is inert, it is still a foreign body and should only be used when the defect is of such size that a sufficient amount of bone cannot be obtained without extensive secondary procedure or when the site of the defect renders it the more suitable material.

HILL.

The Roentgenographic Appearance of Temporomandibular Hydrarthrosis.

Backman, A. L., and Bershon, A. L.: Radiology 46:251-257 (March) 1946.

A case of temporomandibular hydrarthrosis is reported. Twelve radiographs demonstrate changes in the joint over the two-month period of treatment. Technique in the radiographic demonstration is discussed. Literature on effusion into temporomandibular joint is discussed.

JORSTAD.

Roentgenographic Demonstration of Concretions in the Submandibular Glandular Duct by Use of Intraoral Films.

Monardo, G. D.: Radiology 46:53-56 (Jan.) 1946.

Two new positions for demonstrating concretions in Wharton's duct and in the submandibular area are described. Photographs of the patient in proper position, diagrams to show principles of the technique and resultant radiographs of a concretion in the duct illustrate the procedure.

JORSTAD.

Notices

FIRST PAN-AMERICAN CONGRESS OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

OCTOBER 17-19, 1946

Immediately following and overlapping with the annual meeting of the Academy in October of this year, the First Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology will take place, under the sponsorship of the Academy. This Congress will open with a banquet and evening session on Thursday night, October 17, and will continue on the two subsequent days. The first scientific session will be a joint session with the Otolaryngology Section of the Academy, and three additional sessions will take place on Friday afternoon, October 18, Saturday morning and Saturday afternoon, October 19.

All interested members of the Academy are strongly urged to plan on remaining for the Pan-American Congress for which an excellent program is being arranged. The registration fee will be $5.00.

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in Chicago at the Palmer House from October 8 to October 12. Communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

BACK ISSUES OF THE ANNALS

The Annals is desirous of obtaining copies of the following issues now out of print: March, 1943, June 1943, March 1944, September 1944, March 1945, June 1945 and September 1945. $1.50 per copy will be paid for any of these issues returned to us. They should be sent with a memorandum of the sender's name and address to the Annals Publishing Co., 7200 Wydown Blvd., St. Louis 5, Mo.

AMERICAN BOOK CENTER

During the war, the libraries of half the world were destroyed or isolated. There is an urgent need now for the printed materials which are basic to the reconstruction of devastated areas of Europe and the Orient.

The American Book Center is collecting and is shipping abroad scholarly books and periodicals which will be useful in research and necessary in the physical, economic, social and industrial rehabilitation. The Center cannot purchase books and periodicals; it must depend upon gifts from individuals, institutions, and organizations. Each state will be organized to participate in the program through the leadership of a state chairman. Other chairmen will organize interest in the principal subject fields.

What Is Needed. Emphasis is placed upon publications issued during the past decade, upon scholarly books which are important contributions to their fields, upon periodicals (even incomplete volumes) of significance, upon fiction and non-fiction of distinction. All subjects—history, the social sciences, music, fine arts, literature, and especially the sciences and technologies—are wanted.

What Is Not Needed. Textbooks, out-dated monographs, recreational reading, books for children and young people, light fiction, materials of purely local interest, popular magazines, and popular non-fiction of little enduring significance.

How To Ship. All shipments should be sent prepaid via the cheapest means of transportation to The American Book Center, C/O The Library of Congress, Washington 25, D. C. Although the Center hopes that doners will assume the costs of transportation of their materials to Washington, when this is not possible reimbursement will be made upon notification by card or letter of the amount due. The Center cannot accept material which is sent collect. Reimbursement cannot be made for packing or other charges beyond actual transportation. When possible, periodicals should be tied together by volume. It will be helpful if missing issues are noted on incomplete volumes.

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5, Md.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Chicago, Ill., October 13-18, 1946.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland, Ohio
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11, Ill.

American Laryngological Association

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis 8, Mo.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Harry W. Lyman, Carleton Bldg., St. Louis 1, Mo.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

Sections:

Eastern—Chairman: Dr. Robert F. Ridpath, 1737 Chestnut St., Philadelphia 3, Pa.
Southern—Chairman: Dr. Walter T. Hotchkiss, 541 Lincoln Rd., Miami Beach, Fla.
Middle—Chairman: Dr. Oliver E. Van Alyea, 135 S. LaSalle St., Chicago 3, Ill.
Western—Chairman: Dr. Colby Hall, 1136 West 6th St., Los Angeles, Calif.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Meeting: San Francisco, Calif., July 1-5, 1946.

American Otological Society

President: Dr. William E. Grove, 324 E. Wisconsin Ave., Milwaukee 2, Wis.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.

First Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, 3, Pa.
Meeting: Palmer House, Chicago, Ill., October 17-19, 1946.